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A NEW METHOD OF CALCULATING DISCHARGE RATES IN MENTAL DISEASES WITH SPECIAL CONSIDERATION OF THE AGE FACTOR[1]

BY NEIL A. DAYTON, M.D.*

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1. Fundamental Errors in Present Method of Computing Discharge Rates Based upon Cases Discharged per 100 Admissions

IN the 1923 publication of the Bureau of the Census[2] reporting patients in hospitals for mental disease, there appeared a new table in which the discharge rates for the year 1922 were computed. In this table the number of discharges was compared with the number of admissions for the same year, by psychosis. A discharge rate was then calculated per 100 admissions of the same psychosis. As this method apparently had the approval of a governmental agency, it was accepted by other states and is now quite commonly used in statistics on mental diseases.

Over the past few years, however, it has become evident that the Census approach in the matter of calculating discharge rates is not very satisfactory. This is partially due to the overlooking of basic differences between admissions and discharges and also to a lack of consideration of all cases subject to the chance of discharge. In the first place the admissions and discharges of any one year do not comprise the same patients. A study of the outcome of admissions shows that only a certain portion will be discharged and that these discharges will be spread out over many years in the future. Some of the admissions will die while in hospital, some will be deported, others will be transferred, while still others will remain within the institution. On the other hand, the discharges of any one year are the fractional residuals of the admissions that have occurred over many years in the past. The basic incomparability of the two groups and the overlooking of all patients *within hospitals* who are potential candidates for discharge compel serious questioning of the Census method of basing a rate upon the number of admissions and discharges for the same year.

A second point of discrepancy lies in the variation between the proportions of first admissions and readmissions in both admissions and discharges. Under the old method, first and readmissions were combined, the total number of admissions being compared with the total number of discharges. Inspection of the actual differences reveals the danger of comparing such obvious mixtures of classification. For example, in the psychoses with cerebral arteriosclerosis, the discharges (1934) comprised 87 per cent of first admissions and 12 per cent of readmissions. On the other hand, the admissions made up 93 per cent of first admissions and but 6 per cent of readmissions. This factor again emphasizes the incomparability of the two groups, as first admissions and readmissions show widely differing discharge rates.

A third difficulty arises through differences between the ages of admissions and discharges. Over a given period the admissions of any one year tend to sort themselves into four groups in reference to age: (1) those dying; the

1. Read at the 91st annual meeting of the American Psychiatric Association, Washington, D. C., May 13-17, 1935.
From the Division of Statistics and Research, Massachusetts State Department of Mental Diseases. Miss Hariet Dolan, Chief Research Worker, collaborated in the preparation of the statistical material for this paper.

2. Table 42, page 68, Patients in Hospitals for Mental Disease, 1923. Department of Commerce, U. S. Bureau of the Census.

*Dayton, Neil A.—Director Rockefeller Research Project in Mental Diseases and Defect, Massachusetts Department of Mental Diseases. For record and address of author see "This Week's Issue," page 884.

older age groups; (2) those remaining in residence in mental hospitals; the middle age groups; (3) those discharged to the community; the younger age groups; and (4) those transferred, etc.; varied age groups. Thus the discharges of any one year present younger average ages than the admissions of the same year. For example, the psychoses with convulsive disorders (epilepsy) showed an average age of thirty-seven years in first admissions coming into hospitals as contrasted with an average age of thirty-two years in the first admissions discharged during 1934. The readmissions present the same characteristic with an average age of forty-one years in the admissions and an average of but twenty-seven years in the discharges. When we consider that the various ages show widely differing discharge rates we again see the impracticabilty of comparing these two groups.

2. Outline of a New Method of Calculating Discharge Rates in First and Readmissions, by Psychosis and Age

It would seem that the present method of calculating discharge rates not only overlooks certain basic fundamentals but also preserves such obvious mixtures of classification that a satisfactory result cannot be expected. If the old method is not satisfactory, how may we change it? The following is offered for consideration:

Let us begin by determining the entire number of persons within a certain psychosis who are subject to the chance of discharge during a given year. The first step is to divide all cases into first and readmissions, then into specific psychoses, and finally into age groups. The method is outlined in table 1. For purposes of demonstration, this particular table is confined to a single group, the alcoholic psychoses.

In the first line (1) of table 1 we enter the present age of all patients with this psychosis who were resident in mental hospitals at the end of the statistical year. Each patient of this resident group was either in the hospital during the entire year or was admitted or transferred to the hospital during the year. In any case he was subject to the chance of discharge during the year under consideration, and should be included in our basic number. In the second line (2) we enter the patients who were out of hospitals temporarily on visit, parole, etc., at the end of the year, again in accordance with their present age. These patients were either on visit or parole, etc., during the entire year, or were placed out some time during the year. Both groups were subject to discharge and are also included in the basic number. In the third line (3) we list the patients who were discharged during the year, by age at discharge. These cases were either carried over from previous years and discharged, or were both admitted and discharged during the present year. In either case they were under care at some time during the year and subject to discharge. On the fourth line (4) we enter the number of deaths during the year in accordance with the age at death. These patients were either carried over from preceding years and died, or were admitted and died during the present year. The fact that death removed them does not alter the fact that they were subject to the chance of discharge up to the time of death and, there-

TABLE 1

Method of Calculating Discharge Rate Based Upon the Total Number of Cases Under Care, by Age: Alcoholic Psychoses

Source of Cases	Total	Age Groups							
		0-19 Years	20-29 Years	30-39 Years	40-49 Years	50-59 Years	60-69 Years	70-79 Years	80 Years Plus
First Admissions									
1. Resident Population	778	—	14	61	152	253	213	76	9
2. Cases Out (Visits, etc.)	129	—	6	29	45	32	14	2	1
3. Discharges	344	1	27	102	116	67	23	8	—
4. Deaths	69	—	—	10	11	9	28	8	3
5. Total Under Care (1)+(2)+(3)+(4)	1,320	1	47	202	324	361	278	94	13
6. Discharge Rate per 1,000 Under Care (3)÷(5)	261	1,000	574	505	358	186	83	85	—
Readmissions									
1. Resident Population	553	—	2	23	106	170	166	80	6
2. Cases Out (Visits, etc.)	52	—	—	5	14	19	10	4	—
3. Discharges	98	—	4	22	40	24	6	2	—
4. Deaths	26	—	—	—	3	3	5	12	3
5. Total Under Care (1)+(2)+(3)+(4)	729	—	6	50	163	216	187	98	9
6. Discharge Rate per 1,000 Under Care (3)÷(5)	134	—	667	440	245	111	32	20	—

fore, must be included in the basic number. On the fifth line (5) we record the total under care in each age group by adding items (1), the resident population, (2) cases out on visit, parole, etc., (3) discharges during the year, and (4) deaths during the year[3]. This total in each age group shows the basic number of patients under care during the year from all sources and subject to the chance of discharge. In each age group the number of discharges (item 3) is then divided by the number under care (item 5) to produce item (6), the discharge rate per 1,000 cases under care. For example, in the alcoholic psychoses (first admissions) there were 152 cases aged 40-49 years in the resident population, 45 cases who were out on visit, parole, in family care, etc., 116 cases who were discharged, and 11 cases who died during the year. Thus a total of 324 cases with alcoholic psychoses between the ages of 40 and 49 years were under care during the year, and of these, 116 were discharged, giving a discharge rate of 358 per 1,000 under care.[4]

3. Transfers are not considered as admissions or discharges. Separate consideration would be necessary if our statistics were not for the entire state. In reality, transfers have never been removed from the state statistical system. The fact that they are transferred to one institution means that they must have been discharged (by transfer) from another institution on the same day. They were continuously under care throughout the year, and are either reported in the discharges (to the community), in the deaths, or are included in the resident population figure at the end of the year.

4. It is understood, of course, that this method is not mathematically exact in that it does not consider the precise length of time that each patient was under care during the year. It does give a fairly reliable approximation sufficient for our purposes, however. In fact, rates determined by the more detailed method are similar to the rates obtained by our abbreviated method.

Summarizing the results for this particular psychosis, we observe that out of every 1,000 first admissions under care, 261 were discharged during the year. Out of each 1,000 readmissions under care, 134 were discharged. We note higher discharge rates in the younger age groups for both first and readmissions. Discarding the 0-19 and 80 years plus groups because of small numbers involved, the first admissions show the high discharge rate of 574 in the age group 20-29 years, and then drop to the low rate of 83 in the group 60-69 years. The readmissions present the high rate of 667 in the age group 20-29 years, dropping to the low rate of 20 in the group 70-79 years. Both first and readmissions show consistently higher discharge rates in the younger age groups.

3. The New Discharge Rate Applied to the Condensed Classification, by First and Readmissions, Psychosis and Age[5]

Total Discharge Rates, by Psychosis. Table 2 presents the discharge rates per 1,000 cases under care in first and readmissions for the

5. The elaboration of this new method of considering discharge rates is based upon a Condensed Classification of the section on psychiatry of the "New Standard Classified Nomenclature of Diseases". This condensed version was prepared for the statistical committee of the American Psychiatric Association by the writer and, with the detailed classification, was formally adopted at the annual meeting of the Association in May, 1934. It provides a workable classification for statistical purposes and makes possible a continued comparison with the statistics of past years. (See pages 19, 20 and 21 of the "Statistical Manual for the Use of Hospitals for Mental Disease", National Committee for Mental Hygiene, New York, 1934.)

TABLE 2

Discharge Rates for Mental Disorders of the Condensed Classification, 1934, by First and Readmissions: Rates per Thousand Under Care

Mental Disorders	*First Admissions*			*Readmissions*		
	Under Care	Discharges	Discharge Rates	Under Care	Discharges	Discharge Rates
Due to drugs, etc.	57	40	702	24	12	500
Psychoneuroses	314	176	561	127	57	449
With other infectious diseases	50	23	460	11	1	91
Traumatic psychoses	91	30	330	25	5	200
Manic-depressive psychoses	1,557	435	279	1,596	314	197
Due to new growth	18	5	278	2	—	—
Alcoholic psychoses	1,320	344	261	729	98	134
Due to other metabolic diseases, etc.	192	50	260	42	9	214
With organic changes of nervous system	234	57	244	97	21	216
With other disturbances of circulation	84	20	238	10	4	400
With psychopathic personality	175	41	234	135	45	333
Paranoia and paranoid conditions	508	97	191	255	26	102
With epidemic encephalitis	89	15	169	39	4	103
Involutional psychoses	510	71	139	170	19	112
With syphilitic meningo-encephalitis	720	98	136	333	24	72
With other forms of syphilis	128	17	133	58	10	172
Dementia praecox	5,963	609	102	7,934	270	34
With cerebral arteriosclerosis	1,804	169	94	246	24	98
With mental deficiency	1,035	96	93	975	44	45
With convulsive disorders (epilepsy)	833	68	82	359	45	125
Senile psychoses	824	36	44	110	10	91
Undiagnosed psychoses	218	185	849	115	55	478
Without psychoses	1,785	664	372	477	319	669
Primary behavior disorders	62	57	919	17	17	1,000
Total	18,571	3,403	183	13,886	1,433	104

twenty-four groups of the condensed classification. It also records the total number of cases under care and the total cases discharged during 1934. Of each 1,000 first admissions under care during 1934, 183 or about one out of five were discharged. Of each 1,000 readmissions under care, 104 or about one in ten were discharged. The rate for first admissions is nearly twice that of the readmissions.

In the first admissions the psychoses due to drugs show the highest discharge rate of 702. The psychoneuroses are second with a discharge rate of 561. Psychoses with other infectious diseases with 460, and the traumatic psychoses with a rate of 330, are next in order. We then have a group of seven psychoses led by the manic-depressive group which show rates between 279 and 234. Starting with paranoia, we observe a group of six psychoses with discharge rates between 191 and 102, the latter being the rate for dementia praecox. Finally we have discharge rates below 100 occurring in psychoses with cerebral arteriosclerosis (94); psychoses with mental deficiency (93); psychoses with convulsive disorders, epilepsy (82); and the senile psychoses (44). There is a rather remarkable spread between the highest and the lowest discharge rates for 1934. The psychoses due to drugs discharged about seven out of every ten patients under care, while at the other extreme the senile psychoses discharged but one out of every twenty-two patients under care.

Total Discharge Rates, by Age. Table 3 presents the detail of the discharge rates per 1,000 under care for all psychoses together, by first and readmissions and by age. The first admissions present a total discharge rate of 183 per 1,000 for the year. Temporarily discarding the 10-19 and 80 years plus groups because of the small numbers involved, we note that the 20-29 year age group presents the highest discharge rate of 295 cases per 1,000, or about one case discharged in every three of the same age group under care (Graph 1). The age group 30-39 years shows a discharge rate of 227, or about one in four under care; the group 40-49 years a rate of 189 or one in five; the group 50-59 years a rate of 156 or one in six; the age group 60-69 years a rate of ninety-nine or one in ten; and the age group 70-79 years shows the low rate of sixty-three, or one in fifteen. Among first admissions the greater chance of discharge

GRAPH 1.

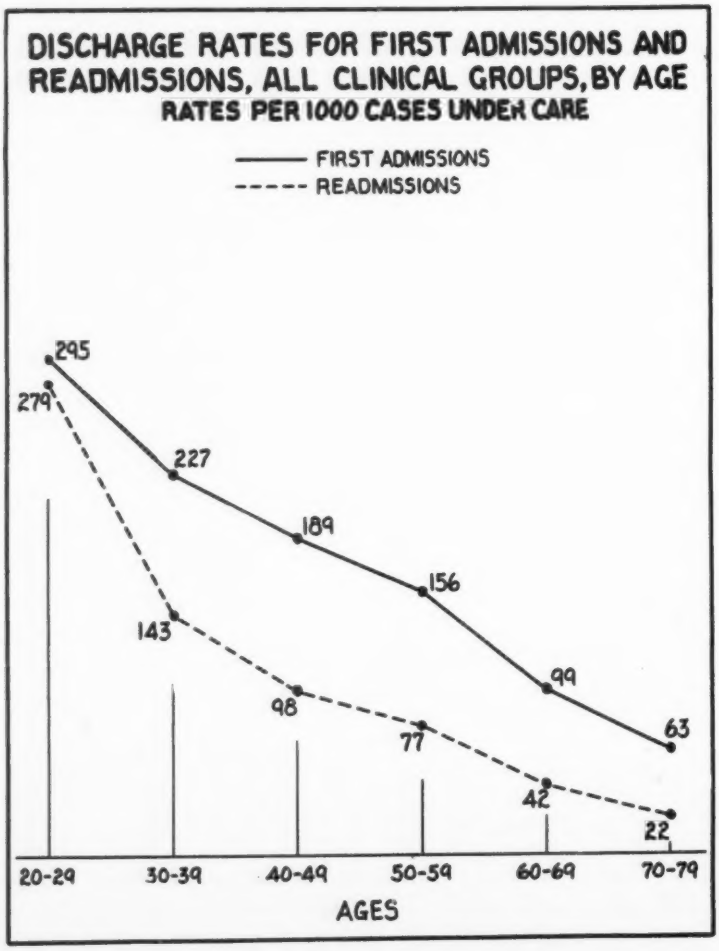

TABLE 3

DISCHARGE RATES PER 1,000 FIRST AND READMISSIONS UNDER CARE[2], 1934, BY PRE

Mental Disorders	Total		10-19 Years		20-29 Years		30-3
	First Admissions	Readmissions	First Admissions	Readmissions	First Admissions	Readmissions	First Admissions
Due to drugs, etc.	702	500	1,000	—	818	1,000	818
Psychoneuroses	561	449	833	600	675	600	578
With other infectious diseases	460	91	400	—	462	1,000	417
Traumatic psychoses	330	200	667	—	571	750	300
Manic-depressive psychoses	279	197	333	579	408	311	310
Due to new growth	278	—	1,000	—	—	—	250
Alcoholic psychoses	261	134	1,000	—	574	667	505
Due to other metabolic diseases, etc.	260	214	600	—	500	333	314
With organic changes of nervous system	244	216	250	600	360	286	250
With other disturbances of circulation	238	400	—	—	—	—	333
With psychopathic personality	234	333	476	636	333	321	15
Paranoia and paranoid conditions	191	102	—	—	833	500	34
With epidemic encephalitis	169	103	133	400	241	100	16
Involutional psychoses	139	112	—	—	—	—	20
With syphilitic meningo-encephalitis	136	72	333	—	45	143	16
With other forms of syphilis	133	172	—	—	—	333	15
Dementia praecox	102	34	308	250	210	151	12
With cerebral arteriosclerosis	94	98	—	—	—	—	25
With mental deficiency	93	45	370	313	136	106	9
With convulsive disorders (epilepsy)	82	125	200	600	141	516	3
Senile psychoses	44	91	—	—	—	—	1,0
Undiagnosed psychoses	849	478	957	667	841	625	87
Without psychoses	372	669	234	821	378	790	3
Primary behavior disorders	919	1,000	923	1,000	1,000	1,000	8
Total rate, all psychoses	183	104	319	599	295	279	2

[2]Cases under care equal cases in residence and cases out of institutions on visit, etc.,

TABLE 3

MISSIONS UNDER CARE[1], 1934, BY PRESENT AGE AND MENTAL DISORDERS OF THE CONDENSED CLASSIFICATION

s	20-29 Years		30-39 Years		40-49 Years		50-59 Years		60-69 Y
	First Admissions	Readmissions	First Admissions	Readmissions	First Admissions	Readmissions	First Admissions	Readmissions	First Admissions
	818	1,000	818	667	750	400	600	571	333
	675	600	578	556	443	366	486	375	292
	462	1,000	417	—	533	—	500	—	—
	571	750	300	250	429	200	190	—	—
	408	311	310	302	276	231	271	182	122
	—	—	250	—	—	—	250	—	500
	574	667	505	440	358	245	186	111	83
	500	333	314	286	324	250	182	214	118
	360	286	250	333	241	43	257	273	148
	—	—	333	—	312	—	333	667	130
	333	321	152	423	276	289	227	412	56
	833	500	340	353	259	98	167	141	84
	241	100	167	111	100	—	167	—	—
	—	—	200	—	231	227	141	159	92
	45	143	162	68	154	74	141	62	54
	—	333	154	167	143	294	179	71	53
	210	151	122	57	69	26	46	18	21
	—	—	250	—	77	167	156	200	114
	136	106	94	71	75	39	45	—	—
	141	516	83	187	63	88	15	27	39
	—	—	1,000	—	333	333	143	286	58
	841	625	878	613	872	542	848	263	800
	378	790	390	634	479	654	507	641	676
	1,000	1,000	800	1,000	1,000	1,000	1,000	1,000	500
	295	279	227	143	189	98	156	77	99

ases out of institutions on visit, etc., on September 30, 1934, plus all discharges and all deaths during the year 1934.

SIFICATION

60-69 Years		70-79 Years		80 Years Plus	
First Admissions	Readmissions	First Admissions	Readmissions	First Admissions	Readmissions
333	400	667	—	—	—
292	200	—	—	—	—
—	—	—	—	—	—
—	—	222	—	—	—
122	104	49	36	167	37
500	—	—	—	—	—
83	32	85	20	—	—
118	125	—	—	—	—
148	—	—	—	—	—
130	500	154	—	—	—
56	—	—	—	—	—
84	58	104	26	—	—
—	—	—	—	—	—
92	52	—	50	—	—
54	91	—	—	—	—
53	100	—	—	—	—
21	5	10	8	—	41
114	122	65	54	68	—
—	—	—	—	—	—
39	—	—	—	—	—
58	138	41	61	24	—
800	—	333	333	—	—
676	487	630	—	1,000	—
500	—	1,000	—	—	—
99	42	63	22	47	86

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is very definitely confined to the younger cases under care.

The readmissions show a total discharge rate of 104 for each 1,000 readmissions under care during the year. The highest rate throughout the various ages occurs in the group 20-29 years with 279 cases discharged, or about one out of every three under care (Graph 1). The readmissions then show a much more precipitate drop in the remaining age groups than is evidenced in the first admissions. The age group 30-39 years shows a rate of 143, or one in seven; 40-49 years a rate of ninety-eight or one in ten; 50-59 years a rate of seventy-seven or one in thirteen; 60-69 years a rate of forty-two or one in twenty-four; and 70-79 years a rate of 22 or one in forty-five. It will be noted that the readmissions present lower discharge rates than first admissions in all ages. In this group we also note definitely higher discharge rates in the younger age groups.

TABLE 4

CASES DISCHARGED FROM NUMBER UNDER CARE AND FIRST AND READMISSIONS, BY AGE: RATIOS

Ages	Cases Discharged from Cases Under Care	
	First Admissions	Readmissions
20-29 Years	1 in 3	1 in 3
30-39 "	1 in 4	1 in 7
40-49 "	1 in 5	1 in 10
50-59 "	1 in 6	1 in 13
60-69 "	1 in 10	1 in 24
70-79 "	1 in 15	1 in 45
Total	1 in 5	1 in 10

To simplify the comparison between first and readmissions table 4 is inserted presenting the ratios for discharges over cases under care in each age group. The first admissions make the best showing in the age group 20-29 years with a ratio of one in three discharged. The ratios increase gradually with the lowest discharge ratio of one in fifteen in the age group 70-79 years. The readmissions also show a very low ratio in the 20-29 year age group, with one out of three discharged. They then show precipitate increases in the ratios to a high of one case in forty-five discharged in the age group 70-79 years. The first admissions make a better showing in all age groups except the 20-29 year group. In the totals, the first admissions discharged one case in every five, while the readmissions discharged but one case in ten[6].

Discharge Rates by Psychosis and Age. We now return to a discussion of the discharge rates within the different age groups for certain psychoses of the condensed classification as outlined in table 3. We have arbitrarily limited these sections to a discussion of the eleven psychoses of the condensed list which had 300 or more cases under care during the year[7]. Dividing those psychoses with less than 300 under care would mean very small numbers in any one age group. Consequently the resulting rates would be more subject to chance fluctuation. We also eliminate consideration of the age groups 10-19 years and 80 years plus as they present even smaller numbers. The eleven psychoses divide themselves into three groups in reference to discharge rates throughout the age groups. These are as follows:

Groups with High Discharge Rates. The first admissions in the psychoneuroses, the manic-depressive and the alcoholic groups, show rather high discharge rates in all age groups (Graph 2). The psychoneuroses show a high discharge rate of 675 in the 20-29 year group, and a low of 292 in the 60-69 year group. The alcoholic psychoses start with a discharge rate of 574 in the 20-29 year group and fall to a low of 83 in the 60-69 year group. The manic-depressive group starts with a high discharge rate of 408 in the 20-29 year group and falls to a low of 49 in the 70-79 year group. The outstanding characteristic of these curves is their rapid fall in rates as the older age groups are approached. The curves for the psychoneuroses and the manic-depressive group are somewhat flatter than that of the alcoholic and tend to preserve fairly high discharge rates for the age groups 20-29, 30-39, 40-49 and 50-59 years. However, in the groups over 60 years of age both show a rapid fall to lower levels. The curve for the alcoholic group shows a more precipitate fall throughout the various age groups.

The readmissions also show rather high discharge rates in practically all age groups (graph 5). Again we note the decidedly higher rates for the younger ages.

6. Owing to space limitations, sex differences are not presented in the tables but are being outlined briefly in this footnote. Among the first admissions the females showed higher discharge rates than the males in eleven clinical groups. These are as follows: with other infectious diseases; traumatic psychoses; alcoholic psychoses; due to other metabolic diseases, etc.; with other disturbances of circulation; with psychopathic personality; with epidemic encephalitis; with syphilitic meningo-encephalitis; dementia praecox; with cerebral arteriosclerosis; and with mental deficiency. In the remaining thirteen groups the males showed higher discharge rates. In the readmissions the females showed higher discharge rates in but five psychoses, namely, with psychopathic personality; paranoia; with epidemic encephalitis; with syphilitic meningo-encephalitis; and in psychoses with mental deficiency. In the remaining nineteen clinical groups the males demonstrated higher rates for discharge. In the totals for all psychoses the males presented a discharge rate of 196 among the first admissions while the females showed a rate of 169. The total readmissions showed a discharge rate of 116 for the males and ninety-one for the females.

The female first admissions present higher total discharge rates in the groups 10-19 years, 20-29 years and 80 plus years, while the males show the higher rates in all age groups from 30 to 79 years. Among the readmissions the females show the higher rates in but two age groups, 30-39 years and 80 years plus, while the males present higher discharge rates in all of the remaining age groups. (For detail see Table 48 of the 1934 Annual Report, Massachusetts State Department of Mental Diseases.)

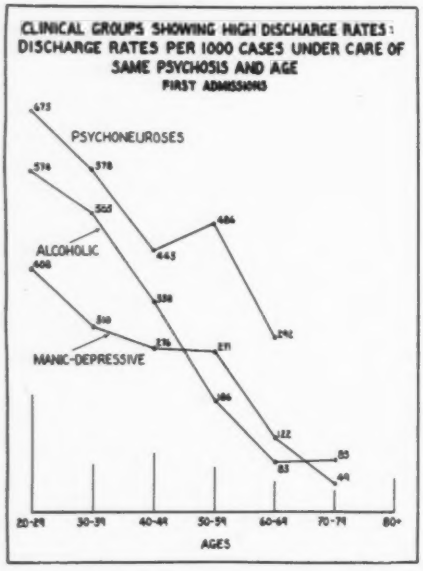
7. Cases under care by psychosis are shown in table 2.

Groups with Intermediate Discharge Rates. Certain psychoses show discharge rates within the various age groups which may be termed intermediate. They are, syphilitic meningo-encephalitis, paranoia, with cerebral arterio-sclerosis, and the involutional and senile groups (graph 3). Among the first admissions the same trend for lower discharge rates in the older age groups is seen. We observe, too, that the rates tend to cluster beginning with the age group 50-59 years, suggesting that age as well as psychosis is an important factor in the matter of discharge. These five psychoses, widely differing from the clinical viewpoint, tend to present the same discharge rate once the cases have passed the age of 50 years. Paranoia and the senile group tend to be more closely associated with age, as we see higher rates in the younger ages and a sharper fall in rates as we approach the older age groups. On the other hand, the curves for syphilitic meningo-encephalitis and the involutional group are flatter and suggest that age is of less importance.

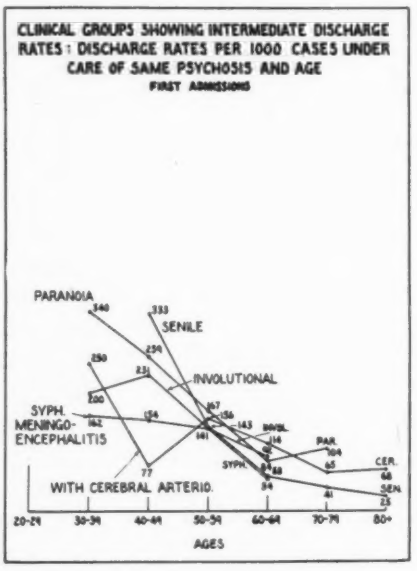
The readmissions of this intermediate group show certain variations, but the same tendency for higher discharge rates in the younger age groups is observed (graph 6).

Groups with Low Discharge Rates. First admissions in the psychoses dementia praecox,

GRAPH 2.


CLINICAL GROUPS SHOWING HIGH DISCHARGE RATES: DISCHARGE RATES PER 1000 CASES UNDER CARE OF SAME PSYCHOSIS AND AGE
FIRST ADMISSIONS
PSYCHONEUROSES
ALCOHOLIC
MANIC-DEPRESSIVE
AGES

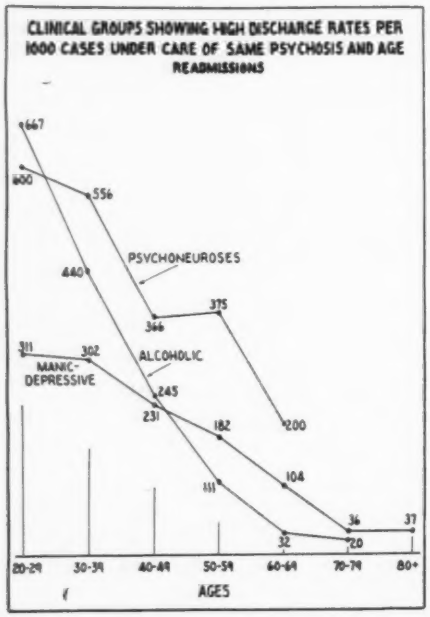
GRAPH 3.



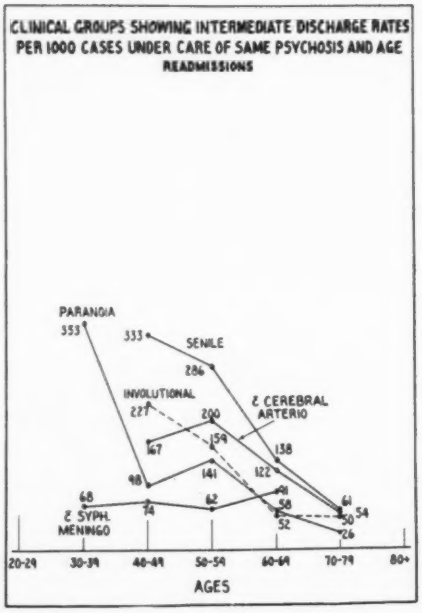
GRAPH 4.

CLINICAL GROUPS SHOWING LOW DISCHARGE RATES: DISCHARGE RATES PER 1000 CASES UNDER CARE OF SAME PSYCHOSIS AND AGE
FIRST ADMISSIONS
DEMENTIA PRAECOX
MENTAL DEFICIENCY
EPILEPSY
AGES

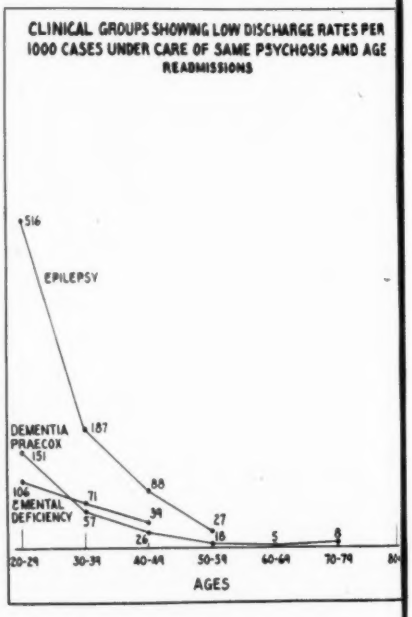
GRAPH 5.



GRAPH 6.



GRAPH 7.


CLINICAL GROUPS SHOWING LOW DISCHARGE RATES PER 1000 CASES UNDER CARE OF SAME PSYCHOSIS AND AGE
READMISSIONS
EPILEPSY
DEMENTIA PRAECOX
C MENTAL DEFICIENCY
AGES

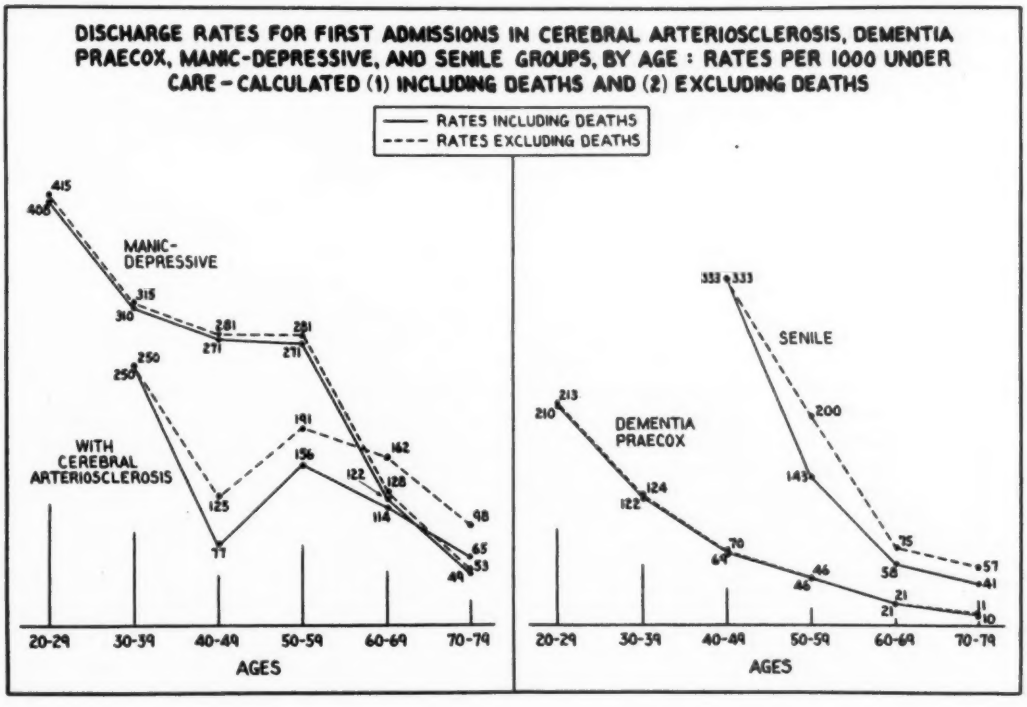
with mental deficiency, and with epilepsy, tend to show low discharge rates in all of the age groups (graph 4). Starting with comparatively low rates in the 20-29 year group they have less opportunity to show any rapid degree of fall in the older ages. However, we note the same general characteristics noted in the other psychoses, namely, higher discharge rates in the younger age groups and lower discharge rates in the older age groups. While the differences appear minor, actually this is not the case. Dementia praecox, for example, discharged one out of five patients in the age group 20-29 years, but only one out of each hundred under care in the age group 70-79 years. Mental deficiency discharged one out of seven patients in the group 20-29 years, but only one out of twenty-two in the group 50-59 years. In reality, marked differences exist here which are not distinctive on the graph because of the low rates in the younger age groups.

In the readmissions, dementia praecox and the psychoses with mental deficiency show uniformly lower discharge rates than the first admissions (graph 7). With a flatter curve and smaller differences between the rates for the younger and older age groups, these readmissions show the least association between the matter of discharge and age. The epileptic psychoses, however, present a marked contrast in showing higher discharge rates in readmissions than in first admissions for all age groups. In the readmissions the rates for this psychosis show an extremely precipitate fall so that the first and readmissions from the age of 50 years onward show only a slight separation.[8]

Possible Sources of Error. When it became evident that lower discharge rates in the older age groups were appearing quite uniformly in all of the psychoses, an effort was made to uncover possible contributing factors other than age alone. One possibility seemed worthy of serious analysis. The very high death rates in the older age groups and the consequent withdrawal of large numbers of cases under care through death, might be expected to produce a lower discharge rate in the older ages. With more patients dying, fewer would be available for discharge. To test this point the discharge rates were calculated *excluding* the deaths. If the deaths were the cause of

8. There is an apparent inconsistency in the placement of some of the psychoses in the groups with high, intermediate and low discharge rates. For example, dementia praecox is placed in the group with low discharge rates, while the senile psychoses are placed in the intermediate group. This apparently disagrees with the data as outlined in table 2 which would place dementia praecox in the intermediate group (total rate 102), and the senile psychoses in the low group (total rate 44). Yet if we compare the distribution of these discharge rates in graphs 3 and 4, first admissions, and graphs 6 and 7, readmissions, we note that the distribution for the senile group is actually higher than that of dementia praecox, both in first and readmissions. This apparent paradox occurs because each of these psychoses will tend to have its total rate approximate the rate for the age group presenting the largest number of cases discharged and under care. Dementia praecox tends to present larger numbers discharged and under care in the younger age groups. Therefore, the total rate for this psychosis will tend to cling to the higher rates observed in the younger age groups. Conversely, the senile group has the largest number of cases discharged and under care in the older age groups. Therefore, the total rate for the senile psychoses will tend to approximate the low rates observed in the older age groups. This shows the advisability of confining comparisons between psychoses to the *same age groups* and to guard against being too greatly influenced by the *total* rates for different psychoses.

GRAPH 8.



diminishing discharge rates in the older age groups, then the calculation of a rate excluding them should produce a flat curve with approximately the same discharge rate in every age group. However, the results for the various age groups were quite uniform with those of the rates *including* deaths, for all psychoses and in both first and readmissions. Graph 8 presents these in four of the psychoses. Two were selected from the point of large numbers involved, dementia praecox and the manic-depressive group. The senile psychoses and the psychoses with cerebral arteriosclerosis were chosen because they involved the older ages with high death rates. Graph 8, in presenting two curves for each psychosis (1) including the deaths and (2) excluding the deaths, shows quite definitely that the deaths have not been a deciding factor in the determination of the trend in discharge rates. In the senile group and the group with cerebral arteriosclerosis the removal of the deaths from cases under care does result in a higher discharge rate, but the two curves in the various age groups remain practically parallel. That is, the resulting discharge rates are higher in the younger as well as the older ages. While the differences are smaller in both dementia praecox and the manic-depressive psychoses, the two discharge rates parallel each other throughout the age groups. It becomes quite clear that the low discharge rates in the older age groups are not to be attributed to the high death rates in those particular ages.

Summary

1. The Census method of calculating discharge rates in mental diseases, based upon the number of cases discharged per 100 admissions of the same psychosis, presents certain inconsistencies which render its use inadvisable. Fundamental errors arising through the lack of separate consideration of first and readmissions, and the disregarding of the age factor, are discussed. These points emphasize the necessity of changing the present method of approach in the matter of determining discharge rates.

2. A new method of calculating discharge rates is presented (table 1). Each psychosis is divided into first admissions and readmissions, and into ten-year age groups. We then determine the number under care in each age group for each psychosis. This is accomplished by adding (1) cases in residence in hospitals at the end of the statistical year, (2) cases temporarily out of hospitals on visit, parole, etc., (3) cases discharged during the year, and (4) cases dying during the year. The resulting total under care is then compared with the total number discharged of the same age, and a rate determined. This new method is applied to each of the twenty-four clinical groupings of the condensed classification, presenting a discharge rate for first and readmissions separately, by age.

3. Of each 1,000 first admissions under care during 1934, 183 were discharged. Of each 1,000 readmissions under care, 104 were discharged (table 2). The total discharge rates on the eleven psychoses presenting more than 300 cases under care during the year are as follows:

Mental Disorders	Discharge Rates Per 1,000 Under Care	
	First Admissions	Readmissions
Psychoneuroses	561	449
Manic-depressive psychoses	279	197
Alcoholic psychoses	261	134
Paranoia and paranoid conditions	191	102
Involutional psychoses	139	112
With syphilitic meningo-encephalitis	136	72
Dementia praecox	102	34
With cerebral arteriosclerosis	94	98
With mental deficiency	93	45
With convulsive disorders (epilepsy)	82	125
Senile psychoses	44	91

4. Considering all psychoses together, the discharge rates in both first and readmissions are very much higher in the younger age groups and decrease as the older age groups are approached (table 3 and graph 1). In the totals about one out of five first admissions under care were discharged and one out of ten readmissions. Eliminating the 10-19 and 80 years plus groups because of the small numbers involved, the rates and ratios are presented in the following table:

Ages	Discharge Rates		Ratios of Cases Discharged from Cases under Care	
	First Admissions	Readmissions	First Admissions	Readmissions
20-29 Years	295	279	1 in 3	1 in 3
30-39 "	227	143	1 in 4	1 in 7
40-49 "	189	98	1 in 5	1 in 10
50-59 "	156	77	1 in 6	1 in 13
60-69 "	99	42	1 in 10	1 in 24
70-79 "	63	22	1 in 15	1 in 45

5. When we study the discharge rates by age groups we find that the psychoses group themselves in three classes presenting, in general, high, intermediate or low rates. All show the same characteristic curve, with higher discharge rates in the younger age groups and lower in the older age groups (table 3 and graphs 2, 3, 4, 5, 6 and 7). The following table is inserted to show these tendencies toward high or low rates for each psychosis ac-

companied by the age group in which the rate occurs. The 10-19 and 80 years plus groups have been eliminated because of small numbers. The figures for the eleven important groups among the first admissions are as follows:

Mental Disorders	High and Low Discharge Rates for Age Groups	
	High	Low
High Discharge Rates		
Psychoneuroses	675 (20-29)	292 (60-69)
Alcoholic psychoses	574 (20-29)	83 (60-69)
Manic-depressive	408 (20-29)	49 (70-79)
Intermediate Discharge Rates		
With syphilitic meningo-encephalitis	162 (30-39)	54 (60-69)
Paranoia and paranoid conditions	833 (20-29)	84 (60-69)
With cerebral arterio-sclerosis	250 (30-39)	65 (70-79)
Involutional psychoses	231 (40-49)	92 (60-69)
Senile psychoses	333 (40-49)	41 (70-79)
Low Discharge Rates		
Dementia praecox	210 (20-29)	10 (70-79)
With mental deficiency	136 (20-29)	45 (50-59)
With convulsive disorders (epilepsy)	141 (20-29)	15 (50-59)

6. To test whether the high death rates in the older age groups were producing the low discharge rates in these ages, the discharge rates were calculated *excluding* deaths. Graph 8 shows the discharge rates both *including* and *excluding* deaths for four psychosis groups. In the senile group and the group with cerebral arteriosclerosis, the removal of the deaths from cases under care results in a slightly higher discharge rate although the trends of the curves remain parallel. In the dementia praecox and manic-depressive psychoses, the discharge rates parallel each other very much more closely. Low discharge rates in the older age groups are not attributable to high death rates in these ages.

7. For many years psychiatrists have been stressing the point that mental disorders should have early recognition and care before they interject a serious handicap into the life of the individual. The findings of this communication support these assumptions in a rather striking way. We see that mental disorders under care in the 20-29 year decade have a much better chance for discharge than cases under care in the older age groups. Even the psychoses occurring in the older ages have this same characteristic, with cases under care in the 50-59 year group showing higher discharge rates than those of the 60-69, 70-79 or 80 plus groups. The younger cases under care have the greater chance of discharge in every psychosis and in both first and readmissions.

HEMOPTYSIS IN TRICHINIASIS*

BY LEONARD J. GOLDWATER, M.D.,† ISRAEL STEINBERG, M.D.,† HARRY MOST, M.D.,† AND JOSEPH E. CONNERY, M.D.†

THE occurrence of pulmonary signs and symptoms in trichiniasis is relatively well known. A great variety of pathological lesions of both lungs and bronchi has been described to account for these findings. Minot and Rackemann[1] in reviewing 102 case histories of trichiniasis to ascertain the frequency of respiratory signs and symptoms quote a number of earlier authors who have written on lung changes in trichiniasis. In Minot and Rackemann's series 50 per cent of the patients showed pulmonary signs or symptoms. There were three cases with blood-tinged sputum, but no case of frank hemoptysis. As early as 1860 Wunderlich[2] reported a case of hemoptysis in a butcher whose illness was at first diagnosed as acute tuberculosis but later, upon recovery, was believed to be trichiniasis. Kestner[3] has described what he calls pneumonia due to trichinae occurring in the fourth week of the infestation. His description of the symptoms strongly suggests infarction. The sputa, according to his report, differ from those of true pneumonia in that they are composed mostly of blackish blood. Hun[4] (1869) described a case of trichiniasis in which blood-streaked sputum occurred about six weeks after the onset of the disease. His description, however, sounds like that of a terminal pneumonia, the sputum being at times rusty. Spink and Augustine[5] more recently in a review of thirty-five cases of trichiniasis observed two patients in whom coughing was productive of bloody sputum.

Askanazy[6] in experimental trichiniasis in rabbits found young trichinae in the lungs causing embolization with hemorrhage into the alveoli and small bronchioles. Frothingham[7] reported an autopsy on a case of trichiniasis which showed many areas of hemorrhage into lung tissue. Trichinellae were found free in the hemorrhage. Thus Askanazy's experimental findings were confirmed in man.

Ordinarily, in the differential diagnosis of hemoptysis, in our opinion, one is not likely to consider trichiniasis as a possibility. In the articles on trichiniasis in several of the commonly used "Systems of Medicine", no men-

*From the Department of Medicine, New York University College of Medicine, and the Third (New York University) Medical Division, Bellevue Hospital.

†Goldwater, Leonard J.—Clinical Assistant Visiting Physician, Third (N. Y. U.) Medical Division, Bellevue Hospital, New York City. Steinberg, Israel—Assistant Visiting Physician, Third (N. Y. U.) Medical Division, Bellevue Hospital, New York City. Most, Harry—Clinical Assistant Visiting Physician, Third (N. Y. U.) Medical Division, Bellevue Hospital, New York City. Connery, Joseph E.—Associate Professor of Clinical Pathology, New York University College of Medicine. For records and addresses of authors see "This Week's Issue," page 884.

tion is made of hemoptysis, nor is it listed in French's or Cabot's Differential Diagnosis. In none of the textbooks of medicine or diseases of the chest which we consulted did we find any mention of blood-spitting in the course of trichiniasis.

Clinical cases of trichiniasis are not uncommon. The number of cases so mild or so atypical that they pass undiagnosed cannot be determined with certainty. In this latter connection the finding by Riley and Scheifley[8] of trichinae in the diaphragms of 18.2 per cent of 520 unselected cadavers and autopsy cases is significant. In view of this apparent frequency of infestation with trichinae, and because of the fact that the symptom of hemoptysis in trichiniasis has not received wide attention, the following cases are being reported.

CASE 1. Patient H. S., white, male, aged thirty-six, nativity U. S., occupation peddler. Admitted to the hospital Feb. 9, 1934. Chief Complaints: Swelling of the face, arms and legs; and loss of appetite.

Previous History: The patient stated that he frequently ate bacon and pork and that recently he had eaten fresh ham.

Present Illness: On the morning of February 1, 1934 the patient was awakened by severe abdominal cramps. These lasted throughout the day. On February 2, 1934 he noticed swelling of his legs from the knees down, and of the arms, eyelids and face. He vomited once on February 3, 1934. Muscular pains appeared on February 5, 1934 and grew worse until the time of admission.

Physical Examination at the time of admission revealed puffiness of the eyelids and engorgement of the conjunctival vessels. The lungs were clear. There was tenderness in both upper quadrants of the abdomen, but the liver and spleen were not palpable. There was tenderness of the triceps and gastrocnemii muscles and pitting edema of the legs up to the knees. The temperature on admission was 99°F., pulse 100 and blood pressure 112/68. The blood count was: R.B.C. 2,800,000, Hb. 9 grams per 100 cc., W.B.C. 7,800, polymorphonuclear leukocytes (young) 7 per cent, (mature) 41 per cent, lymphocytes 16 per cent, eosinophiles 36 per cent. The urine showed nothing abnormal at this or any other time, nor did the stools.

Course: A biopsy of the left deltoid muscle done on February 14, 1934, revealed the presence of typical trichinellae. On February 20, 1934, the patient developed pain in the left chest, which was relieved by adhesive strapping. On February 22, 1934 the entire right leg became swollen and tender. On the following day the patient began to raise sputum heavily streaked with blood and containing clot-like masses of blood and muco-pus. For seven days the patient continued to raise this type of bloody sputum. By March 5, 1934 the swelling of the right leg had subsided. A chest x-ray taken at this time revealed a flattening of the left diaphragm with obliteration of the left costophrenic sinus, and a small patch of parenchymal infiltration in the fifth left interspace. Several days later the left leg showed changes similar to those in the right, indicating at first phlebitis and later thrombosis of the femoral vein. This ultimately subsided, and no further complications arose.

In this case, the diagnosis of trichiniasis was definitely established by the finding of trichinellae in a muscle biopsy specimen. In the following two cases the parasites were not demonstrated, but the history, clinical course, and laboratory findings were such that there seemed to be little doubt that trichiniasis was the diagnosis.

CASE 2. Patient H. C., white, male, aged twenty-four, nativity U. S., occupation medical student. Admitted to the hospital November 7, 1933.

Chief Complaints: Headache, malaise, muscular pains, swelling of the eyelids.

Previous History: There was no recollection of the ingestion of raw or poorly cooked pork, and no diarrhea.

Present Illness: On November 2, 1933 upon awakening in the morning the patient noted edema of both upper eyelids, especially the left. On November 4, 1933 generalized malaise was experienced. The edema of the eyelids became more marked. On the same day muscle and joint pains developed. Diplopia, lasting from three to five minutes, was experienced on November 4, 1933 and again on November 5, 1933. Severe frontal headache began on November 5, 1933. Hoarseness with cough and moderate mucoid expectoration developed on November 6, 1933. The patient was seen at the student health office during this time but could not be prevailed upon to enter the hospital until the fifth day of his illness.

Physical examination at the time of admission to the hospital revealed nothing significant except tenderness over the left maxillary sinus, and a slight mucopurulent exudate on the posterior pharyngeal wall. The temperature was 101.8° F., pulse 106 and blood pressure 128/82. The blood count on admission was as follows: R.B.C. 4,490,000, Hb. 11 grams per 100 cc., W.B.C. 9,500, polymorphonuclear leukocytes (mature) 33 per cent, (young) 10%, lymphocytes 7 per cent, monocytes 1 per cent, eosinophiles 44 per cent. The urine at no time showed any abnormality.

Course: On November 8, 1933 the temperature rose to 104.4° but later fell to 103°. The following day pterygia were present in both eyes and the spleen was palpable. The patient was hoarse at this time. A skin test with trichinella antigen was strongly positive. On November 10, 1933 the temperature was lower, but there were muscular soreness and tenderness in the extremities. During the next seven days there was gradual improvement. On November 17, 1933 the white cell count was 14,500 with 41 per cent eosinophiles and three days later it was 17,000 with 38 per cent eosinophiles. At this time (November 20, 1933) the patient complained of severe pain and marked tenderness in the right subscapular region. There had been no chill and there was no cough. The skin over the right side of the chest posteriorly was hyperesthetic, and there were diminished expansion and decreased breath sounds over the right lung. The pain was relieved by strapping the chest. At no time was there any evidence of consolidation. On November 21, 1933 and November 22, 1933 on several occasions the patient raised small amounts of tenacious mucoid sputum mixed with bright red blood. No trichinae were found in this sputum. On November 23, 1933 pain similar to that just described developed on the left side. There was no significant rise in temperature accompanying either of these attacks of pain. The course from this point on was uneventful and the patient was discharged on December 3, 1933.

CASE 3. Patient S. S., white, male, aged thirty-seven, occupation butcher, nativity Austria. Admitted February 27, 1934.

Chief Complaint: Cough accompanied by pain in the chest, and bloody sputum.

Family History: Irrelevant.

Previous History: Irrelevant except that patient stated that he had "bronchitis" for fifteen years.

Present Illness: On February 2, 1934 patient developed what he called a cold, with coryza, chills, fever, malaise and nonproductive cough. There was a transient attack of nausea and diarrhea. After several days, weakness, backache and pains in the extremities developed, confining the patient to bed for a week. He was treated by his local physician for "grippe" and improved somewhat, but there persisted a dull pain in the chest, weakness, occasional feeling of chilliness associated with sweating, and a cough productive of tenacious mucoid sputum. He returned to work but on February 23, 1934 he coughed up about two drams of blood. The following night he raised a similar amount of blood. Subsequently the amount of blood decreased so that when he entered the hospital there was merely a blood-streaked sputum.

Physical Examination on admission revealed nothing abnormal except for numerous sibilant and sonorous râles over both lungs and a few faint, moist râles at the left base and in the left axilla. There was some dulness at the left base.

Laboratory data: Hb. 12.2 grams per 100 cc., R.B.C. 4,100,000, W.B.C. 8,000, polymorphonuclear leukocytes (young) 4 per cent, (mature) 59 per cent, lymphocytes 37 per cent, Wassermann negative. Sputum on two occasions negative for acid-fast bacilli, but was definitely bloody. The temperature was 100° F. at the time of admission but subsequently was never above normal. Chest roentgenograms on February 28, 1934 and March 3, 1934 revealed changes associated with chronic bronchitis and emphysema. Bronchial neoplasm or bronchiectasis was suspected, but bronchoscopy and lipiodol studies revealed nothing abnormal, and the patient was discharged.

After the patient returned to his home, additional history was obtained by a member of his family who is a physician. It was learned that the patient had eaten poorly cooked pork immediately before the onset of his illness, and that he had had edema of the eyelids for several days before he entered the hospital. Further blood studies revealed on several occasions an eosinophilia of 20 per cent to 30 per cent.

COMMENT

It is interesting that in all three cases hemoptysis occurred approximately three weeks after the onset of symptoms of trichiniasis. This is in accord with the results of Askanazy[6] who found trichinae in the lungs of infected animals no sooner than three weeks after experimental infestation. In cases 2 and 3 there is no explanation for the hemoptysis except that proposed by Askanazy, namely, embolization due to trichinae. In case 1, however, the presence of femoral thrombophlebitis offers another possible etiology for the bloody sputum, although there is no reason to believe that the mechanism in this case was different from that in cases 2 and 3.

The failure to find trichinae in the sputum is consistent with the observations of other workers[9].

SUMMARY

1. Three cases of trichiniasis with hemoptysis are reported.
2. In one case the diagnosis was missed because of failure to demonstrate eosinophilia and failure to obtain a complete history.
3. In two cases, in which the sputum was examined, no trichinae were found.
4. The importance of considering trichiniasis in the differential diagnosis of hemoptysis is emphasized.

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UNCINARIASIS AND APPENDICITIS*

BY M. K. KING, M.D.†

DURING the past eighteen months we have had the privilege of seeing in this hospital a large number of young adults from a section of the country where hookworm, Necator americanus, is quite prevalent. In a number of these cases, presenting fairly characteristic signs and symptoms of acute appendicitis, the appendix was found at operation to be not sufficiently pathological to account for the clinical picture. This observation was especially frequent in those patients infested with hookworms. In an attempt to find a relationship between uncinariasis and this syndrome resembling acute appendicitis, a study was made of 100 cases treated in this hospital for suspected appendicitis.

In the extensive literature dealing with uncinariasis and appendicitis separately, there has been little intimation of any connection between the two.

*From the U. S. Marine Hospital, Savannah, Georgia.

†King, M. K.—In charge of Surgical Service, United States Marine Hospital, Savannah, Georgia. For record and address of author see "This Week's Issue," page 884.

McGehee[1] in 1910 reported a case of acute appendicitis in which hookworms were found in the lumen of the appendix; the hookworms apparently playing an etiological rôle in the attack.

Giudice[2] in 1913 reported a case in which a perforated appendix was found to contain seven ankylostomes. The pathology in the appendix was apparently initiated by the hookworms.

Risquez[3] in 1917 reported twenty-eight appendices removed from Venezuelan subjects and examined for parasites. The ova of Necator americanus were found in five, the larva in one; but no adult hookworms were found.

Verdelet and Sigalis[4] in 1921 reported a case of acute appendicitis in which large numbers of hookworm ova, but no worms, were found in the appendix.

Bacigalupo[5] in 1930 reported a study for parasites made on 100 surgically removed appendices, in which no hookworms were found.

Gordon[6] in 1932 reported a study for parasites made on 26,051 surgically removed appendices. Oxyurids were found in 311, amebae in four, tapeworm in one, and roundworms in three cases. No uncinaria were found. The material for this study was presumably obtained in the north central portion of the United States however, and therefore out of the hookworm belt.

In the present study 100 cases were taken consecutively as they were sent to this hospital with suspected acute appendicitis. The patients were all enlisted members of Civilian Conservation Corps located in the neighborhood of this city. Almost without exception the cases infested with hookworms were young men who had been reared in nearby rural communities.

TABLE 1

100 Consecutive Cases of Suspected Acute Appendicitis

	Cases	Per Cent
Total	100	
Suspected appendicitis, acute	100	100%
Uncinariasis	50	50%
Appendectomy	68	68%
Treated without operation	32	32%
Pathological appendix	51	75%
Normal appendix	17	17%
Hookworms found in appendix	0	
Recovered	100	100%

Of this group of 100 patients sixty-eight (68 per cent) were treated surgically for acute appendicitis. The remaining thirty-two (32 per cent) were treated conservatively, with apparent recovery. Hookworm ova were found in the stools of fifty patients (50 per cent). The percentage of infestation would probably have been higher had not a number of these patients received treatment for hookworms prior to entering the hospital. Thirty-eight of the sixty-eight cases which were treated surgically were infested with hookworms (55.9 per cent).

After removal at operation each appendix in this group was submitted to the laboratory where a careful pathological examination was made. No evidence of uncinaria was found in any of the sixty-eight appendices examined, and seventeen were reported as non-pathological. Forty-one ranged through all grades of inflammatory changes from mild to fulminating types.

Each of the seventeen cases in which the appendix was found to be normal presented a fairly convincing picture of acute appendicitis prior to operation. The following case is fairly typical of this group:

T. H., a previously healthy young male adult, was taken ill with an acute abdominal pain. The pain localized in the right lower quadrant and did not radiate. There was no diarrhea. He vomited several times before admission to the hospital. Acute tenderness and some rigidity were present on pressure over the right lower abdomen. The temperature was 37.2°C.; the white blood count was 11,600, neutrophiles 73 per cent, eosinophiles 5 per cent. Hookworm ova were present in the stool. At operation the whole small intestine and cecum were acutely inflamed, thickened and edematous. No other pathology was present in the abdomen. The appendix was removed and when examined by the pathologist was reported to be normal. Convalescence following operation was uneventful.

The most striking feature of this case was the acute inflammation in the small intestine and cecum. This was an almost constant finding in those patients with uncinariasis however. The character and intensity of the inflammation varied. The whole small intestine was frequently of a fiery red color, thickened and edematous, but with relatively little peritoneal involvement; the inflammatory process being apparently confined to the mucosa and muscularis. In two cases the inflammation was present in the form of localized patches, ranging from one to three cm. in diameter, scattered profusely throughout the jejunum. These patches showed clearly through the visceral peritoneum, which was but little involved. In no case did the inflammation extend beyond the cecum; but the cecum was frequently involved.

"Mainly in the jejunum but discernible as high as the stomach and as low as the colon, the wall of the intestine in heavily infested cases is chronically inflamed, with a heavy outpouring of thick, rarely blood-stained mucus. The worms are so effectually imbedded in this that without deliberate search many will be missed. The mucosa in heavily infested cases may show atrophy." (Lane[7].)

Uncinariasis was present in twelve of the seventeen cases (70 per cent) in which the appendices were found to be normal at operation. Each of these twelve cases with uncinariasis presented the characteristic inflammation of the

small intestine and cecum described above. The clinical picture preceding operation in each case was that of acute appendicitis. After a careful review we are unable to find a single case treated with appendectomy in which the findings did not appear to warrant the operation. We feel that the hookworms must have been responsible for the abdominal pain and tenderness; we have no explanation for the acuteness of the onset, and the severity and localization of the pain. It appears that in patients with uncinariasis we may have a syndrome so characteristic of acute appendicitis as to be difficult or impossible of preoperative differentiation. This syndrome has probably occurred more frequently than reports would indicate, in sections of the country where uncinariasis is prevalent.

FIG. 1. "The head end of Necator americanus attached to the intestinal mucosa. The effects of the powerful suction produced by the strong oesophagus are evident." ("Hookworm Infection," Lane.)

It must not be construed from what has been said that there is justification for delaying operation in an apparent case of acute appendicitis because of the presence of hookworm ova in the stool. In twenty-six of our cases both diseases were present. The patients affected with both frequently showed no more profound symptoms than those in which only uncinariasis was present and the appendix normal. The following case represents a type which is frequently seen.

J. U., a previously healthy young male adult, was taken ill with a moderately severe abdominal pain, which localized in the right lower quadrant. He vomited several times before admission to the hospital. His bowels had not moved since the onset of the attack. He was moderately tender to pressure over the right lower abdomen, with some muscular rigidity present. The admission temperature was 37.2 degrees C. The white blood cell count was 9,200, neutrophiles 59 per cent, eosinophiles 1 per cent. Hookworm ova were present in the stool. At operation the appendix was found to be gangrenous throughout, well walled off, and not perforated. The whole small intestine and cecum were fiery red and swollen. No free exudate was present in the general peritoneal cavity. There were no hookworms found in the lumen or walls of the appendix. Following operation the patient progressed to an uneventful recovery.

The preoperative picture in this case was not striking, and yet the pathology was advanced. Whether there is some relationship between the pathology in the appendix and the hookworms in the small intestine is an interesting question. Having been unable to find the worms in a single instance in the appendix, we are unable to conclude that they are a common factor in the etiology of acute appendicitis. However it appears entirely possible that an acute inflammation produced by hookworms in the small intestine and cecum might include the appendix, where the anatomy of this organ would predispose to the process becoming fulminant.

The treatment given for uncinariasis to the patients in this series consisted of 3 cc. of carbon tetrachloride, preceded and followed by saline purgation. In surgical cases the treatment was given when the patient had recovered from operation, usually about the tenth postoperative day; in others it was given only after acute abdominal symptoms had subsided. There were none other than mild reactions from the drug. It was occasionally necessary to repeat the treatment before a stool free from ova could be obtained.

SUMMARY

1. Uncinariasis may at times give rise to a syndrome closely simulating acute appendicitis.

The differential diagnosis is difficult or impossible preoperatively.

2. In patients with uncinariasis the small intestine and cecum are frequently found to be acutely inflamed at operation. The appendix may or may not be involved. The inflammation involves chiefly the mucosa and muscularis leaving the peritoneum relatively clear.

3. There is little evidence that hookworms are a common cause of acute appendicitis. No hookworms were found in sixty-eight appendices removed surgically from patients coming from a highly infested hookworm district; in thirty-eight of these patients hookworm ova were found in the stool. It is conceivable, however, that a widespread intestinal inflammation caused by hookworms might involve the appendix, where the process, due to the anatomy of this organ, might become fulminating.

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CATARACTS AND DINITROPHENOL*

BY DAVID G. COGAN, M.D.,† AND FRANCES C. COGAN, M.D.†

DURING the past two and one half months, there have occurred in the literature no less than twenty cases of cataracts developing after the use of dinitrophenol. This sudden and somewhat tardy appearance of the entity is striking in view of the widespread use of the drug over the past several years. The physiological action of the drug is fairly well understood, and the type of cataract is remarkably constant (vide infra). With this in mind, it is our intention to review the reported cases and to suggest a possible pathogenesis for dinitrophenol cataracts.

SURVEY OF CASES

Age. Average age of occurrence was 37.9 years with extremes of 25 and 50. Even the oldest member in this group is younger than the age at which we find the usual senile cataract.

Sex. With one exception (No. 13), all cases occurred in women. This is undoubtedly due to the relatively greater consumption of dinitrophenol among women.

Dosage. Here we wish to make an extremely significant observation, and one of great importance to the dispensing physician. Excepting one case only (No. 11), all were taking dinitrophenol in the recommended therapeutic doses and the majority were taking it under the supervision of a physician. The average maximal daily dose of the patients in this series was .45 grams which is not large when one considers the weight of most of the patients taking the drug. Furthermore, there were three patients, developing cataracts, who took a daily dose which at no time exceeded .3 grams (No. 12, No. 14, No. 17).

Time of taking dinitrophenol. The total period during which dinitrophenol was taken was stated definitely in sixteen of the twenty cases. It averaged 8.4 months with a minimum of one month (No. 14)‡ and a maximum of 21 months (No. 7).

Loss of weight. By some this is taken to be proportional to the efficacy of the drug and hence is recorded here. Total loss of weight was noted in twelve of the twenty cases. It averaged 50¾ pounds over an indefinite period. Minimum weight loss was 21¼ pounds and maximum was 100 pounds.

Other toxic reactions. In only three of the twenty cases is there any mention of other toxic manifestations. Of these three, one (No. 20) had merely slight gastrointestinal upsets when she took the drug, one (No. 10) complained that her feet and hands became numb, and one (No. 13) had a frank peripheral neuritis.

Rate of cataract development. It is impossible to give any average for this group but a glance at the table will show the characteristic rapidity with which the cataracts develop, once that they are first noticed. Thus, in at least one case (No. 1), vision declined from the first observable dimness to mere light perception, all in the short space of a single week. In five more cases virtual blindness ensued within a month (No. 2, No. 3, No. 7, No. 8, No. 13).

Type of cataract. This is probably the most significant from the ophthalmologists' point of view for two reasons: first, the cataracts have a characteristic morphology; secondly, cataracts of this type point to some exogenous etiology. In ten of the twenty case reports, there is adequate de-

*Massachusetts Eye and Ear Infirmary, September 19, 1935.

†Cogan, David G.—Clinical Assistant in Ophthalmology, Massachusetts Eye and Ear Infirmary. For records and addresses of authors see "This Week's Issue," page 884.

‡There is some question of this patient having taken dinitrophenol for a short time one year previously. In this case, the minimal time of taking dinitrophenol would have been a three-month period (No. 13).

Author	Age	Sex	Av. Dosage in Gms.	Total Time Taken	Gms. Total Dosage	Lbs. Loss of Weight	Other Toxic Reactions	Mo. Cataract Developed After 1st Dose	
1. Boardman	50	♀	—	1 yr., 8 mo.	—	76½	None reported	19	1 wk light
2. Boardman	36	♀	.1-.5 p. diem	10 mo.	—	52½	None reported	16	1 mo light
3. Boardman	39	♀	— Under M.D.	8-9 mo.	—	"Satisfactory"	None reported	—	30 da light
4. Boardman	40	♀	— Not under M.D.	Several mo.	—	—	—	—	Rapid
5. Boardman	—	♀	—	—	—	—	—	—	Rapid
6. Boardman	—	♀	—	—	—	—	—	—	Rapid
7. Horner, Jones, Boardman	50	♀	.1-.5 p. diem	21 mo.	107.2	75	None reported	20	1 wk. 0.1
8. Horner, Jones, Boardman	36	♀	Up to .5 p. diem	16 mo.	114.1	66½ Later 12	None reported	16	3½ wk light
9. Horner, Jones, Boardman	30	♀		3 4/5 mo.	316	21¼	None reported	15	Visio tion)
10. Shutes	44	♀	18 p. mo.	4 mo.	72	55	Numbness of hands and feet	12	1 mo. and 2
11. Shutes	39	♀	25 p. mo.	5 mo.	135	—	None reported	12	3 mo. tingu
12. Shutes	36	♀	9 p. mo.	4 mo.	36	35	None reported	12	2 mo and 2
13. Cogan, Cogan	38	♂	.2-.5 p. diem	3 mo.	—	100	Peripheral neuritis	12	2 wks
14. Cogan, Cogan	25	♀	.3 p. diem	? 1 mo.	9	—	None	? 4	3-4 w perce
15. Barber*	27	♀	.3 p. diem	3-6 mo.	—	15	None	9	7 wks to 1/2
16. Man*	27	♀	10.4 p. mo.	5 mo.	52.5	None	None	16	2 mo finger
17. Lazar	44	♀	7.6 p. mo.	12 mo.	91.25	None	None	6	Less 0.2
18. Gifford†	42	♀	—	—	—	2 per wk. for few mo.	None reported	14	In les light
19. Kniskern	37	♀	16.08 per mo.	11½ mo.	133	—	None reported	14	
20. Allen, Benson	38	♀	12.16 per mo.	12 mo.	146	32	Slight gastro-intestinal upset	24	Less to cou

*To be published.
†Quoted by Lazar.

aract oped er ose	Rate of Cataract Development	Type of Cataract
	1 wk. from dimness of vision to light perception	Suggestive of "toxic or metabolic disturbance"
	1 mo. from incipient cataract to light perception	—
	30 days from dimness of vision to light perception	—
	Rapid	—
	Rapid	—
	Rapid	—
	1 wk. from vision of 0.6 and 0.8 to 0.1	Subcapsular opacities in posterior cortex resemble "brass filings"
	3½ wks. from vision of 0.8 R. E. to light perception R. E.	Subcapsular opacities in posterior cortex resemble "brass filings"
	Vision 0.8 O. D. (only observation)	Fine subcapsular opacities in posterior cortex resemble "brass filings"
	1 mo. from "blurry" vision to 4/200 and 2/200	Posterior cortical and subcapsular opacities
	3 mo. from "blurry" vision to distinguishing objects only	—
	2 mo. from failing vision to 2/200 and 20/70	Silvery cholesterin appearance to opacities
	2 wks. from 20/20 to 20/200	Cataracta complicata
	3-4 wks. failing vision O. S. to light perception O. S. O. D. 20/30	O. S. complete clouding of lens O. D. clouding along sutures
	7 wks. from vision of 20/70 and 20/30 to 1/200 and 3/200	Diffuse clouding with no vacuoles or clear spaces
	2 mo. vision reduced to counting fingers and 16/200	Diffuse grey opacities in posterior cortex
	Less than 4 mo. vision reduced to 0.2	Subcapsular opacities with saucer shaped coppery appearance of posterior cortex
	In less than 1 yr. vision reduced to light perception	Intumescent cataract
	—	—
	Less than 4 mo. from failing vision to counting of fingers at 1 ft.	Fine granular subcapsular opacities with appearance of "hammered copper" on posterior surface

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scription of the lens changes to recognize the type as "Cataracta Complicata." In the remaining ten, the descriptions are insufficient to tell just what the nature of the lens changes is, but we may infer they were of the same type. The characteristics, par excellence, of Cataracta Complicata are the subcapsular position of the opacities, and the initial involvement of the posterior cortex. This is aptly described as having a "brass filing" appearance. In cataracts of this type, the ophthalmologist suspects a source of systemic intoxication.

With the data listed in the accompanying table we have attempted to correlate the time at which the cataracts appeared following the first ingestion of the drug with: (1) the total dosage taken; (2) the total time during which the drug was taken; (3) the average rate of dosage per month; and (4) the loss of weight. The relations have been computed on a scale varying from + 1.00, which represents a 100 per cent direct correlation, to —1.00, which represents a corresponding inverse correlation.

The total dosage and time of appearance of cataracts have a relation of +.26. This has only slight statistical value in a small series. We might expect a correlation of this order inasmuch as the total dosage will be greater, the later the cataracts develop. The total time of drug ingestion and time of appearance of cataracts have a relation of +.58 which has a somewhat higher statistical value. But here again, the longer it takes for the cataracts to appear, the more time will there have been to take the drug. It is doubtful if these two factors have any further causal relationship. It should be noted here, however, that, with the exception of one case (No. 14), the minimum time between first taking the drug and appearance of cataracts was six months. The one exception we exclude because of its alleged unreliability. The average time between first taking the drug and appearance of cataracts was slightly more than fifteen months. It is because of this delayed effect that cases are only now beginning to appear although the drug has been used for several years. For the same reason we may expect many more cases in the near future.

The relation between the time of appearance of cataracts, both with the rate of dosage and with the loss of weight approximate a statistical value of —.12 and +.17. Neither of these figures is large enough, positively or negatively, to be significant in a small series.

As a result of these studies, therefore, we cannot say that the production of cataracts by dinitrophenol is a function of the total amount taken, nor of the length of time that it was taken, nor of the rate with which it was taken, nor of the loss of weight.

COMMENT

The above points to an individual susceptibility as the significant factor in the development of dinitrophenol cataracts. Other toxic manifestations of the drug show a similar variability. This, however, is not a unique characteristic of the drug as the allied chemical, trinitrotoluol (T.N.T.), has also shown a varying susceptibility among ammunition workers.

We have had the opportunity to examine nine other patients who had been taking small doses of the drug, over a period of forty-nine days, none of whom showed lens changes or any notable toxic reactions. Others[2] have made a similar observation.

The modus generendis of these cataracts presents an interesting problem. The drug, dinitrophenol, is chemically and physiologically allied to the naphthol group, especially dinitro-alphanaphthol. Edsall[8] states that the two drugs have similar metabolic and toxic effects, which vary widely for individuals of the same species. The chief difference between the two is quantitative only. It may be of significant interest, therefore, that cataracts have been produced in both industrial naphthol poisoning and in experimental rabbits, and that these cataracts, as in this series, developed in the short period of a few weeks. However, there are, to date, two incompatibilities in this analogy. In the first place, we have not been able to produce dinitrophenol cataracts in our limited experiments on rabbits*; secondly, there is no report of retinal or iris lesions in these cases as have been noted to accompany naphthol cataracts. We cannot say at present, therefore, that the cataracts of dinitrophenol have the same pathogenesis as those of naphthol poisoning.

On the other hand, the tissue anoxemia produced by dinitrophenol may account for much of the damage. Dinitrophenol apparently increases the cellular metabolism to a degree inordinate with its oxygen supply. The tissues become acidotic, lactic acid is piled up, and the products of decomposition are incompletely oxidized. That this is largely responsible for the toxicity of dinitrophenol is shown by the marked reduction of its toxic effects through the inhalation of pure oxygen[9]. There is no reason why the lens epithelium should not partake of this general tissue anoxemia and be thereby damaged. In fact, it is our opinion that the relatively remote position of the lens from its nutritive source renders it more vulnerable than other tissues.

In view of the morphology of the lens changes and the known effects of dinitrophenol on tissue metabolism, it seems likely, as we have noted in a previous publication[4], that

*We have been giving large doses of dinitrophenol to two rabbits for a period of two months without the development of cataracts.

the cataracts are the result of damage to the lens epithelium rather than to precipitation of the lens constituents. This is why the toxic effects occur, not as immediate reactions, but after more or less prolonged use of the drug, and may occur after the drug has been discontinued altogether. Four cases in this series (No. 9, No. 12, No. 13, No. 20) developed cataracts practically a year after the discontinuance of the drug while two more cases (No. 4, No. 15) developed them, four and three months after it was discontinued. Furthermore, we have immersed immature cataractous lenses (removed intracapsularly) in a 2 per cent solution of dinitrophenol without the development of more opacities as we might expect were it a primary precipitation of the lens substance. We conclude, therefore, that the anoxemia of the lens epithelium resulting from dinitrophenol is, in all probability, responsible for the cataracts.

SUMMARY

The entity of dinitrophenol cataracts appears to be established by the number of recent reports. The cataracts have a characteristic morphology and rate of development. A statistical study of the relationship of time of cataract formation with (1) total dosage, (2) total time of taking drug, (3) average rate of dosage, and (4) loss of weight, shows no definitely causal connection. Like other toxic manifestations of the drug, the development of cataracts shows a bizarre idiosyncrasy but unlike most other toxic reactions, cataracts develop from merely therapeutic doses. The pathogenesis of cataract formation may be similar to that of the allied drug, naphthol, but seems more likely a result of tissue anoxemia with consequent damage to the lens epithelium.

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ELECTROSURGICAL APPENDECTOMY*

BY LESTER R. WHITAKER, M.D.†

AMONG surgeons there is considerable difference of opinion with regard to inversion of the stump of an appendix. The opponents hold that when the stump is tied and inverted it is simply buried in the wall of the cecum; that this leads inevitably to inflammatory changes in the wall, and possibly to abscess. They maintain that perforation of the gut during the insertion of the purse-string suture may lead to infection; and finally that the whole process may result in extensive adhesions around the cecum.

On the other hand, the proponents of inversion hold that any abscess that forms in the wall after the inversion will quickly slough into the lumen of the bowel, especially if the stump of the appendix has been crushed and a tie of fine plain catgut used. And they say further that any capable surgeon should be able to insert a purse-string suture without perforating the gut. They hold that when not inverted inflammatory reaction in the stump may result in adhesions of the small intestine to the appendiceal site; or, that, since the circulation is shut off distal to the tie, sloughing may result in leakage.

Still others assume that the whole discussion is "much ado about nothing", and that good results are obtained by either method. There is one point, however, to be brought up here. If a surgeon should nick a hole in the colon during an operation, would he pick up the tissues and close the opening with a catgut ligature, cauterizing the stump thus created; or would he place inverting stitches? There is little difference between this situation and that with a dangling stump of an appendix. It is generally agreed, however, that with a gangrenous appendix and indurated cecum inversion should not be attempted, but adequate drainage employed.

In the past well-conceived methods have been used to avoid abscess of the cecal wall following inversion of the appendiceal stump. One of the best is that of Lexer[1]. For several years a slight modification of this method has been used at the Massachusetts Memorial Hospitals. A brief description follows: With linen or catgut suture material a stitch is taken just distal to the base of the appendix on the wall of the cecum, and a tie made, the object being to secure a small vessel usually present at this point. Then, running a continuous Lembert stitch, picking up the cecum on either side of the base of the appendix, a cuff is built around the appendix for about a half-inch, the sutures being

*From the Evans Memorial for Clinical Research and Preventive Medicine and the Surgical Clinic of the Massachusetts Memorial Hospitals.

†Whitaker, Lester R.—First Assistant Visiting Surgeon, Massachusetts Memorial Hospitals, Boston. For record and address of author see "This Week's Issue," page 884.

so placed as to pull the cecal wall rather tightly around the appendix. The base of the appendix is crushed and the clamp applied. The continuous suture is loosened, but held ready to be pulled up. Then the appendix is cut off with a knife below the clamp, and the stump of the appendix retracts as the suture is tightened. One or two sutures are taken over the top, completing the operation.

Obviously, this method is open to criticism on the ground that contamination may occur as the stump of the appendix retracts. Cautery excision would tend to prevent this. To place a ligature around the stump before it is pulled in would defeat the object of the whole procedure and amount to the same thing as inversion with a purse-string suture. In the absence of the tie, however, there is sometimes considerable hemorrhage into the lumen of the colon.

The question of inversion of the stump of the appendix, as against the method just described (Lexer), has been investigated by Bürkle-de la Camp[1]. This author found on monkeys that, when the stump was tied and inverted, there was pronounced exudation between the stump and the overlying serosa, with considerable leucocytic infiltration and inflammation of the cecal wall. The Lexer technique, however, showed no such disadvantage.

Electrosurgical appendectomy by the method to be described avoids the danger of contamination and hemorrhage, and at the same time prevents formation of an abscess in the cecal wall about the inverted stump.

The method follows: After cutting away the mesentery, the base of the appendix is crushed over a distance of about a half-inch, down to and, perhaps, including a small portion of the cecal wall. The pressure should not be vigorous enough to crack through and produce contamination. The crushed mucosa and fecal contents of the lumen are pushed out by squeezing with the clamp, which is discarded. A tie of coarse linen is placed around the base of the appendix in the crushed zone; that is, a small devitalized section is left below the linen tie. Then a clamp is applied about a half-inch distal to the tie. With the "cutting current", heavy dehydration, low voltage, and using a needle electrode, the appendix is cut off. The cutting is done with the side of the needle, progressing slowly, perhaps moving the needle from side to side a short distance to make it act more as an ordinary cautery, to seal off the lumen as it is cut across, and to sterilize the fecal contents. Then with the "coagulating current", using biterminal fulguration (sparking), the whole stump is thoroughly treated, including the small crushed portion of the cecum *below* the linen tie. The devitalized stump is then inverted with a double row of Lembert sutures. A closure as secure as would be required with a hole in the bowel is necessary.

Experimentation upon dogs has shown that the stump sloughs off into the cecum within twenty-four hours, leaving the walls nicely inverted and approximated at this point.

In three patients who died after major abdominal operations where incidental appendectomy was performed by this method, the cecum was examined postmortem. In one, after forty-eight hours, the stump had sloughed into the lumen of the bowel leaving the inverted edges closely approximated. In the second, also after forty-eight hours, the stump was hanging by a few small threads of necrotic tissue, and the closure of the cecum underneath it was firm. In the third, after four days, the edges of the infolded gut were clean.

Examination of the records of fifty personal cases of electrosurgical appendectomy reveals good results. The method was used only in those cases in which there was no induration of the cecum itself. The average stay in bed was eight and one-half days. There were no untoward symptoms, nor any complications referable to the abdomen.

The advantages of electrosurgical appendectomy as described are as follows: It is simple and aseptic; it promotes rapid sloughing of the stump into the lumen of the colon, without danger of hemorrhage or abscess of the cecal wall; and it leaves a firm closure of undamaged tissues.

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PRIMARY ABSCESS OF THE OMENTUM*

BY RALPH W. FRENCH, M.D.†

THE great omentum is a structure to which are ascribed multiple functions, yet a discussion of its pathology rarely appears in the literature. Von Recklinghausen and Bizzozzero[1] as early as 1885 by experimentation with animals demonstrated the major rôle of the omentum to be the absorption of substances from the peritoneal cavity. When the omentum was removed, there was marked increase in susceptibility to abdominal infection. If the circulation of a particular organ was impaired, as in the spleen or kidney, the omentum formed a capsule over the necrotic organ and walled it off, retarding the spread of infection. By the formation of adhesions the omentum walled off inflammatory processes and lessened the es-

*Read before the Newport Medical Society, February 12, 1935.

†French, Ralph W.—Surgeon, Truesdale Hospital, Fall River, Mass. For record and address of author see "This Week's Issue," page 884.

cape of intestinal contents in cases of rupture of a viscus. It furnished protection and warmth and filled in irregularities between intestinal coils. Arnaud[2] ascribes to the omentum the following functions:

"It fixes microscopic bodies, surrounds large immobile foreign bodies, has secretory powers, regulates isotonicity and maintains the sterility of the peritoneal cavity. It has greater power of exudation and fibrin production than the peritoneum."

It is, therefore, something of a paradox when the omentum, which is known to possess these bactericidal and protective powers, itself becomes inflamed or infected. Experience with a recent case of inflammation of the omentum which went on to abscess formation led to a search of the literature for similar cases. Few reports of primary abscess of the omentum were available, but those found presented interesting hypotheses as to the etiology and altogether showed a wide range of pathology.

The condition, it seems, of primary acute epiploitis or inflammation of the omentum was first observed by Championnière[3] in 1892. He encountered two cases in a series of 275 herniotomies in which a portion of the omentum was found in an old hernial sac at the time of operation or became inflamed shortly afterwards. (The French and German authors always refer to the condition as epiploitis and never use the word omentitis in their descriptions of this entity.)

Schnitzler[4] collected twenty-four cases including his own, Braun[5] added eight others, and Zesas[6] collected twelve more, so that by 1909 the total number of cases reported was forty-four. Since this time about thirty more have appeared in the literature.

Chronic abscess of the omentum may not be so rare as these statistics seem to indicate. It is difficult to determine the exact pathology at operation when one simply drains a localized abscess of the abdomen adherent to the abdominal wall. The abscess in such cases is generally considered secondary and there is no opportunity to locate the origin of the inflammatory process. The cases recorded, however, are sufficiently numerous to compare the various theories offered in answer to the question, "Why should the omentum fall victim to infection?"

There is controversial opinion as to the mobility of the omentum and its property of migrating to areas of inflammation. The most plausible explanation is offered by Schutz[7]. After experimenting with dogs he found that peritoneal irritation caused hyperemia, and as the blood vessels of the omentum lost their tortuosity, there was a blind mass movement of the entire omentum toward the focus of irritation. Hertzler suggests a chemotactic attraction between the area of irritation and leucocytes in the infiltrated omentum. Some authorities claim that only the edge of the omentum has the power to migrate, slowly drawing the omentum to wall off an infected area. When the omental edge has been removed in animals, the power to migrate seems to have been lost.

For the most part, the anomaly of omentitis is ascribed to operations or to the rise of inflammatory processes within the abdomen. It is affirmed that ligation of the omental flaps with silk or catgut can cause inflammation of the omentum. After an operation for, let us say, inguinal hernia, a portion of the omentum might accidentally have been tied off; if the patient experiences abdominal pain with rise of temperature, it is natural to infer that infection resulting from a break in asepsis or from the ligature material is the cause of symptoms. The site of the inflammatory mass is usually in the region where the omentum was tied off. Proof of this condition is borne out by the fact that a piece of catgut was found in the abscess cavity in several instances.

From a study of his eight cases Braun draws the following conclusions:

"These inflammatory tumors of the omentum . . . appear at a period varying from one to ten weeks after operations in which the omentum is tied off; there are recorded cases in which the interval was as long as four months and even three years. It is natural to infer that infection, either previously existing or resulting from the ligature material, is the cause. The swellings may be found almost anywhere in the lower abdomen depending partly upon the locality in which the omentum was tied off; usually, however, they are in the vicinity of the umbilicus. The surface is smooth, the mass is firm in consistency, sensitive to pressure, and is not influenced in its position by respiratory movements. If there are no parietal adhesions, the swelling is movable laterally and upward but not downward; as the intestines lie behind it, there is dullness on percussion. Usually pain is the first evidence followed by fever, occasionally chills and vomiting; the course of the temperature depends upon whether or not suppuration takes place."

As a preventive measure, Braun cautioned us to ligate only non-inflamed omentum and to include only small portions of the omentum in each ligature. This theory of etiology is all the more plausible because of the fact that the pus in some cases with abscess has contained shreds of silk as well as catgut. Hamann[8] added three cases attributable to this cause.

Steiger[9] mentioned a case in which a stab wound in the abdomen caused infection of the omentum from perforation of the intestine; Sowles[10], Giraud[11], and Labry and Arnulf[12] each reported a case in which the inflammation of the omentum followed as the result of a blow in the abdomen. After resection of the inflamed area the patients recovered.

It is well established that the omentum may also become inflamed by contact with an inflamed viscus or as a result of operative trauma. After operating upon three patients with hy-

pertrophic inflamed omentum, Emerson[13] came to the following conclusion:

"The abnormal condition of the omentum at the site of pain and the relief after partial omentectomy warrant the conclusion that there is a distinct entity as a late sequela of the pelvic surgery, which I call chronic localized omentitis."

These three patients after pelvic surgery complained of severe abdominal pain and excruciating tenderness with fullness and resistance in the pelvis suggestive of tumor. When the abdomen was explored the only lesion found was the hypertrophic omentum.

Symptoms often make their appearance after appendectomy, cholecystectomy, or other pelvic operations, and in these cases operative soiling cannot be ruled out, especially when the pain is localized at the site of the previous operation. Arnold[14], Thévenard[15], and others reported cases of postoperative inflammation of the omentum in which, although the omentum had not been disturbed during operation, the patient did not make a complete recovery and symptoms of inflammation appeared from one month to one year afterwards.

There are some cases, however, which must be placed in another category; those in which there has been no previous operation. In these the origin of the condition must be considered embolic, since the inflammatory tumor is apparently not connected with any demonstrable intraperitoneal pathology at the time of exploration. The only pathology in the abdomen is that found in the omentum where the inflammatory process appears to be primary. In Adams's case[16] there was no previous operation or injury, and the only recent illness had been pneumonia one year previously and frequent colds. This patient complained of acute pain in the lower right abdomen for four days. At operation an abscess of the omentum was found overlying a normal appendix and cecum.

Pauchet[17] reported two cases which he interpreted as acute exacerbation of a chronic omentitis. The omentum showed inflammatory change at the lower border. The omentum was resected and a normal appendix removed incidentally with recovery in both cases. He stated that the epiploön in this type of case became infected by way of the lymphatics.

In 1929 Schomberg[18] reported three cases of acute hemorrhagic epiploitis. A section of the omentum was discovered to be dark red, densely infiltrated, and there was considerable serosanguineous fluid in the abdomen. Two of the patients had always been in good health, the third man had epididymitis several years before the onset of symptoms of epiploitis. In these three cases the appendix was normal. After resection of the omentum the three patients recovered.

In McWhorter's[19] three cases the condition in the first apparently followed severe bronchitis; in the second it was probably due to an infected thrombus from appendicitis; and in the third no etiology was found. Walther[20] reported several cases in which there was no pathology except the inflamed omentum in the peritoneal cavity. He pointed out, however, that it might be argued that the original pathology subsided and the omentum detached itself from the old lesion.

Thorek[21] divided cases of omental suppuration into two groups: (1) the Schnitzler-Braun or chronic type, and (2) the Küttner-Schmieden or fulminating type. Boltanski[22] and Rouslacroix[23] also reported cases as either congestive inflammatory or chronic adhesive. Thorek ascribed an embolic origin to the acute inflammatory omentitis and believed that both types might occur with or without adhesions to neighboring viscera.

It appears from Haller's[24] research that the omentum is found altered in patches in many laparotomies; thus he reported 372 patients treated for epiploitis. In many of these cases, however, the degree of inflammation was slight. Instances were few in which the condition simulated malignant disease and in these the prognosis was discouraging.

In Pantzer's two cases[25] the condition followed an operation in which convalescence was retarded by peritonitis, and he interpreted the pathology thus:

"The *materiae morbi* passed the prohibitory portals of the omentum, became implanted there at various points and were held in inoffensive isolation for a long time. Then owing to faulty metabolism or debility as a result of severe physical strain, the poisonous elements in these foci were liberated and caused grave systemic sepsis."

These patients did not get well after operation and remained semi-invalids complaining of abdominal tenderness and pain, yet between attacks showed no evidence of disease. At operation the omentum was found studded with numerous minute abscesses and the structure was thickened in the areas in which the abscesses were located. Pantzer believed that in these two cases the products of inflammatory reaction were held in retention in the omental tissue, contrary to the usual process whereby the infected emboli are gradually taken up by the lymph channels of the omentum and eventually disappear. In the milder cases, the portion of omentum which has become adherent to an inflamed viscus when stripped off shows little evidence of inflammation within itself.

Once the omentum becomes infected, the process seems to take one of two courses: either the omentum becomes more and more hypertrophied or multiple tiny abscess cavities form in scattered areas of the thickened and inflamed omentum. Of all the cases reviewed, Pantzer's two fatal cases of multiple abscesses of the omentum and Maunsell's[26] report of chronic fibrous epiploitis, in which the omentum

was transformed into a smooth, pearl-white membrane adherent deep down in the pelvis, are examples of the two extremes of this pathology of the omentum.

The case which I encountered recently and which prompted a study of the cases reported in the literature was one of acute primary inflammation of the omentum that went on to abscess formation.

The patient was a woman forty-six years old, who was admitted to the Truesdale Hospital October 25, 1934, complaining of pain and soreness in the left lower quadrant of the abdomen which had persisted for six weeks. Two years previously she was in the hospital for bronchopneumonia complicated by anemia. At that time it was noted that the spleen was slightly enlarged. Shortly afterwards she returned for cholecystectomy for gall stones and her convalescence was very satisfactory, except that it was complicated by a small ischiorectal abscess. The condition cleared up readily, however, and the patient remained well for a year and eight months until the onset of her present illness.

On admission the patient gave a history of moderate abdominal pain for two weeks in the left lower quadrant gradually increasing in severity. A large, hard, smooth, tender mass was palpable in the left lower quadrant of the abdomen. Her temperature was 100.4 degrees, pulse 100 and respirations 24. The red blood count was 4,420,000, white count 9,400, and hemoglobin 60 per cent. The nonprotein nitrogen was 30.6 mgm., the blood sugar 100 mgm. The blood Wassermann was negative. The general physical examination was otherwise not remarkable. A diagnosis of ovarian cyst with twisted pedicle was made and the patient prepared for operation.

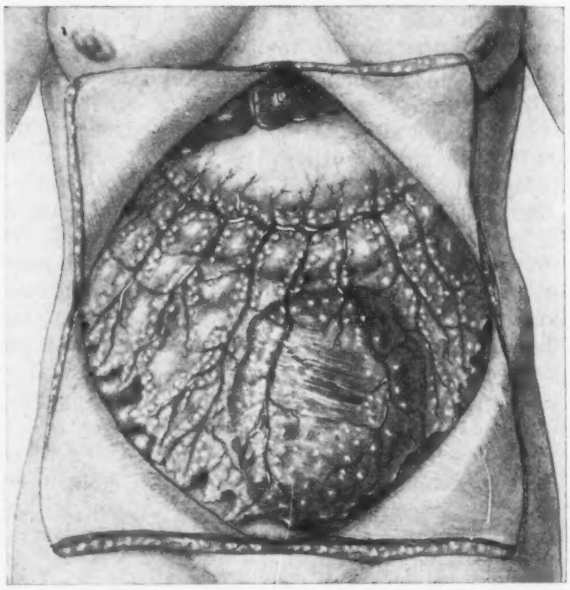
Exploration revealed an inflamed omentum like a mat, six inches in diameter, in the left lower abdomen and pelvis. The center was broken down and contained thick staphylococcus pus in a cavity two inches in diameter. Around this the omentum was very hard and fully two inches thick. (See illustration.) It was easily lifted up from the pelvic organs and intestines which it covered and showed no adhesions to these organs. It involved approximately the left half of the omentum, whereas the right half was apparently normal and free. There were only a few filmy adhesions under the scar of her gall bladder operation. They did not appear to play any part in this picture. From the omental edge the induration extended upward to the transverse colon. Because of the extensive suppuration and its proximity to the colon, it was deemed safer to drain the abscess cavity than to remove the cakelike mass.

Since the process was so hard that I thought it simulated malignancy, a piece was removed for microscopical examination. The pathologist reported an acute exacerbation superimposed on a chronic inflammation.

The pelvic organs were all found to be normal and the sigmoid and small intestines under the mass showed no sign of inflammation or other abnormality.

Her convalescence was slow. She complained of general abdominal discomfort and a substernal burning sensation, and slept poorly. There was a cramplike pain in the upper abdomen after the ingestion of food which persisted for nearly two weeks following operation. On the tenth postoperative day the white count was still 21,700. Drainage, which was profuse at first, gradually diminished but the patient continued to complain of tenderness in the left lower quadrant, and the temperature remained elevated around 102 degrees.

Five weeks after the first operation a secondary abscess developed and pointed to the left and above the center of the previous abscess. Her temperature continued at 102 degrees and the white count varied from 21,700 to 11,200. This abscess was opened through a counterincision. There was a soft area of necrotic tissue in the thickened omentum into which drains were placed. Thereafter her



Primary abscess of the omentum.

recovery was steady with a gradual drop in the temperature and white count to normal ten days after the second incision was made. After this second operation the mass palpable beneath the abdominal wall became slowly but steadily smaller. The patient left the hospital nearly six weeks after the first operation. She has remained well with no inflammatory symptoms and is leading a normal active life.

In this case drainage of the abscess cavity effected a cure. According to Hamann,

"If the progressive increase in the size of the swelling, fever, parietal adhesions and leukocytosis indicate the presence of pus, an incision is to be made and the abscess cavity evacuated and drained. The ultimate results are in the vast majority of cases favorable."

Drennan[27], however, disagrees and states:

"Unless there is abscess formation, the outlook usually is good. In the presence of abscesses, however, a less favorable prognosis must be given, for these abscesses may break, thus causing a fatal peritonitis."

He advises laparotomy in every instance where abscess formation is suspected. Whenever it is feasible, extensive resection has been the method of choice when induration and abscess formation are present.

COMMENT

Cases of acute primary omentitis coexistent with pancreatitis, sigmoiditis, and diverticulitis have been reported by Baur[28], Hatzieganu[29], Lepoutre[30], and Canonne[31]. An acutely inflamed omentum gave rise to encapsulating peritonitis in two cases reported by d'Allaines and Jomain[32], and was the cause of intermittent fever of long duration in several instances[33]. Thus the omentum, whatever may be its pathology, is a structure to be reckoned with in complications of the abdomen and may be the sole cause of symptoms. The differential diagnosis is difficult because acute primary omentitis may simulate many other inflammations such as acute appendicitis, cholecystitis, ovarian cyst with twisted pedicle, or diverticulitis. As Schrager pointed out:

"If the mass is at or above the level of the umbilicus, there must be differentiation from pathology of the appendix or the gall bladder, wandering liver, neoplasms of the spleen, or tumor of the colon; if the mass is below the umbilicus, one must rule out ovarian cysts, or inflammatory masses or tumors of the pelvis."

Furthermore, the condition must be differentiated from other pathological conditions of the omentum itself, such as simple torsion, lipoma, cysts, malignancy, tuberculosis, or syphilis.

In the severe cases the prognosis is discouraging, whether treatment is conservative or daring, but if operation is performed fairly early in the course of the disease, the usual outcome is recovery.

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THE ROLE OF PERIPHERAL CIRCULATORY FAILURE IN CLINICAL MEDICINE*

BY DANA W. ATCHLEY, M.D.†

IN addressing the New England Heart Association on the subject of circulatory failure my New England conscience forces me to state that I am not a cardiologist and that although my interest in the shock syndrome has included many phases of the problem my actual investigative work has been confined only to those aspects which are related to electrolyte physiology. If one is interested in this ubiquitous complication of disease, there is a fascinating literature available for his consumption. The whole gamut of man's absurdity and man's brilliance in the study of natural phenomena is included in the various writings about shock. One may see oft-recurring the almost obsessive urge to invent a foreign toxin to explain a simple physiological process. The so-called "toxemias" of burns, diabetic coma, pyloric obstruction, Addison's disease and traumatic shock are examples of this misguided ingenuity. On the other hand, in few fields have the obvious truths been so clearly stated and so long

*From the Department of Medicine, College of Physicians and Surgeons, Columbia University, and the Presbyterian Hospital, New York City.

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†Atchley, Dana W.—Associate Visiting Physician, Presbyterian Hospital, New York City. For record and address of author see "This Week's Issue," page 884.

buried. I shall point out to you that about seventy years intervened between the first rational analysis of shock in cholera including the appropriate therapeutic suggestions and its general practical application. Janeway pointed out clearly, in 1907, the more frequent termination of severe infectious diseases by peripheral vascular failure than by cardiac failure, yet most modern textbooks of medicine still place their emphasis on the use of cardiac stimulants in such situations.

However, a large number of painstaking investigations, many of them remarkable in their vision far beyond contemporary scientific boundaries, have brought to this field a knowledge of the underlying disturbances of physiology that may produce the shock syndrome and have, moreover, clarified in great detail the effects of shock itself upon the organism. As one reviews the research of these workers, it is apparent that there are a few simple mechanisms which constantly recur and that the appearance of this complication in a wide variety of totally different diseases may be correlated with one or more of these mechanisms. Thus are we able to understand the apparent identity of the final stages of such totally different diseases as cholera, diabetes and severe burns.

For purposes of simplification it may be stated that whenever it occurs, the state of shock is the physiological result of an acute disparity between the circulating blood volume and the functioning capacity of the vascular bed. This result can, of course, be brought about by a relatively rapid decrease in blood volume or by a sudden expansion of the vascular bed, and also by a concurrent appearance of both changes. When this discrepancy reaches an advanced stage its ill effects upon the individual are profound and indeed often fatal. I need offer only brief reminder of the clinical picture with its evident great prostration, cold extremities, sweating, pinched features, pallor, sunken eyes, small rapid pulse and low blood pressure. As the result of varied researches, many of them from the Harvard physiological laboratories, the pathological physiology is well understood. There is stasis of blood in the capillaries with a generalized anoxemia and tissue asphyxia. Before the blood pressure actually drops the cardiac output in most instances decreases, thus augmenting the process. The capillaries dilate due to oxygen want, with consequent increase in the vascular bed and leakage of serum into the tissues. Dissipation of serum into the tissue spaces contracts the blood volume and increases blood viscosity. A vicious circle is thus initiated that rapidly hastens the downward course. The kidneys cease to serve their purpose in relation to acid base and electrolyte physiology. Nitrogenous waste products accumulate. If infection be present there is serious interference with the immunity mechanism. Bacterial toxins cannot be normally neutralized or removed. The whole body economy is at a standstill during what is usually a very critical period because of the original disease process of which the circulatory failure is a complication. However, it is not alone the seriousness and frequency of shock in clinical medicine that demands attention but also and, perhaps more important, the fact that it is in many instances an avoidable danger. In order to prevent shock or treat it properly when it occurs, it is necessary to be familiar with the various routes by which the final stage may be reached. In no realm of clinical medicine is theoretical physiology more fruitfully productive of practical therapy than in this.

The mechanisms that produce a discrepancy between the blood volume and the vascular bed are in ultimate analysis few and simple. A decrease in blood volume may be due to direct loss of whole blood as in hemorrhage, it may be due to loss of salt and water from the blood by a variety of routes and, finally, it may be due to loss of serum from capillaries that have dilated and hence become more permeable to protein. The vascular bed may be increased in size by toxins that cause the capillaries to dilate either by direct physiological effect or by poisoning them. Vasodilatation may also be produced by the neurogenic route, through the sympathetic nervous system.

These underlying mechanisms offer an excellent framework for a discussion of the occurrence of shock in clinical medicine. Because it is the first competent analysis of such an underlying mechanism that I have been able to discover, I would like to detail to you a letter that Dr. W. B. O'Shaughnessy of Newcastle-upon-Tyne wrote in 1831 to the *London Medical Gazette*. This letter embodied the results of several years of what he termed his "experimental inquiries" into the cholera. His terse summary of a physiologic mechanism in terms of modern chemistry is a delightful contrast to the usual clinical sophistry of that day. As I quote his conclusions, I am sure you will agree that his work would still be modern 100 years after he lived. He states:

"1. The blood drawn in the worst cases of the cholera, is unchanged in its anatomical or globular structure.

"2. It has lost a large proportion of its water, 1000 parts of cholera serum having but the average of 850 parts of water.

"3. It has lost also a great proportion of its neutral saline ingredients.

"4. Of the free alkali contained in healthy serum, not a particle is present in some cholera cases, and barely a trace in others.

"5. Urea exists in the cases where suppression of urine has been a marked symptom.

"6. All the salts deficient in the blood, especially the alkali or carbonate of soda, are present in large quantities in the peculiar white dejected matters."

Quite wisely, I believe, a contemporary reviewer of Dr. O'Shaughnessy's "Chemical Pathology of Cholera" called particular attention, in *The Lancet* of 1832, to the "strictly logical series of deductions, disfigured neither by empty hypothesis, nor by untenable and wild speculations."

In other words, by quantitative chemical analysis, he demonstrated dehydration, salt depletion, acidosis and urea retention and discovered that the base loss occurred via the copious stools.

Turning next to the therapeutic implications which naturally follow from the biochemical disturbances found in the disease, Dr. O'Shaughnessy states further that the cure is seemingly dependent upon two principles: "First, to restore the blood to its natural specific gravity (i.e., its water content); secondly, to restore its deficient saline matters." He then states that, "The first of these can only be affected by absorption, by imbibition, or by the injection of aqueous fluid into the veins. The same remarks, with sufficiently obvious modifications, apply to the second." The practical application of these principles was described as follows: "In severer cases copious enemata of warm water, holding the natural salts of the blood in solution are strongly recommended." When absorption is entirely suspended the author recommends the injection into the veins of tepid water holding a solution of the normal salts of the blood. Thus it is apparent that over one hundred years ago a physician with an investigative and critical mind was able, with the crude methods at his disposal, to unravel the mechanism of the essential manifestations of cholera, viz., dehydration, salt depletion and shock. Furthermore, he logically employed the specific therapy indicated by such a disturbance in physiology.

Although the implications of O'Shaughnessy's extraordinary work were confirmed by a number of workers in Britain, as well as on the Continent, the struggle to overcome the traditional prejudices of those in authority is beautifully exempified by the following words written by Dr. J. Pidduck to the *London Medical Gazette,* August 21, 1832.

"Turning hopelessly away from the Central Board and Local Hospital, I resolved to pursue the experiment (i.e., saline therapy) among the poor in my district, convinced that the brandy and laudanum system had been too highly recommended, and too long sanctioned by authority, to admit of the introduction by the same individuals of another system so diametrically opposed to it. Perhaps such a revolution in a cherished opinion, and a favourite practice, would be a stretch of candour and liberality almost superhuman."

In spite of the fact that in 1850, the German biochemist Karl Schmidt repeated and greatly amplified these analytical results of O'Shaughnessy, there were subsequently but occasional references to the therapeutic value of intravenous salt solutions. It was not until Rogers, and Nichols and Andrews in 1909, used saline injections with a remarkable decrease in the mortality of a cholera epidemic in the Philippine Islands that this rational treatment became generally accepted.

I have taken time to trace the development of the chemical pathology of cholera because it presents, as I have already said, one of the earliest instances in which there was an understanding recognition of the physiologic processes involved in the development of this common complication of many disease conditions, viz., dehydration, salt depletion and shock. Furthermore, it was in the treatment of cholera that, for the first time, a rational replacement therapy was instituted with, I might add, the anticipated clinical benefit.

Chronologically, the next correct appraisal of this problem of dehydration may be found in the Guy's Hospital reports of 1874. Dr. C. Hilton Fagge described, therein, "A Case of Diabetic Coma, treated with partial success by the injection of a saline solution into the blood." The most impressive feature of this pioneer experiment in therapy was the rationale which he offered. It is best expressed in his own words, "What suggested to my mind the advisability of injecting a saline solution into the blood in this case was the idea that coma was due to the drain of water from the system, caused by the diabetes. I suppose that the hypothesis upon which I acted was essentially similar to that which formed the basis of the like treatment in the collapse of cholera." The importance of this observation was nine years later overshadowed by Stadelmann's discovery of the existence of an acidosis in diabetic coma. Stadelmann quite logically felt that the replacement of alkali was the obvious point of attack. This concept so completely dominated the treatment of diabetic coma that even the more modern textbooks omit mention of the clear correlation between dehydration and circulatory collapse which Fagge had demonstrated in 1874. One must not neglect to state that for at least twenty-five years salt solution has been routinely employed in the treatment of diabetic acidosis. However, it was usually done with the coincident assumption that the picture of low blood pressure, thready pulse, oliguria and collapse resulted from "toxic" cardiac failure rather than from shock due to decreased circulating blood volume and vasomotor paralysis. We were able to show in our clinic that the loss of salts and water in

diabetes is dependent upon two apparently unrelated mechanisms. One of these is the obviously necessary loss of base resulting from the excretion of salts of the ketone acids. The other is more difficult to understand but it is definitely associated with the occurrence of severe glycosuria, and may be found even in the absence of ketosis. These two forces working together in an acute diabetic crisis bring about a loss of salts and water that is cataclysmic in its effects upon the patient. It is amazing to note the failure of the textbooks to emphasize this predominant pathological force in diabetic coma; it is not an exaggeration to assert that aside from wholly unrelated complications such as pneumonia, the chief cause of death in severe diabetic acidosis is shock, a death which may occur with normal blood sugar and bicarbonate levels. I belabor the point because it is usually easy to prevent such deaths.

The clinical picture dependent upon dehydration, salt loss and consequent shock is found in many other disease conditions. Prominent among these is the group characterized by pyloric or high intestinal obstruction, as well as those patients in whom for some reason there exist fistulous openings from the upper gastrointestinal tract or the bile ducts. It naturally follows that the picture of shock in these patients results in part at least from constant vomiting or persistent drainage of body fluids normally rich in sodium salts. It should be pointed out that in certain cases of intestinal obstruction no actual loss of salt from the body occurs, but the same result is effected by the outpouring of salts and water into the distended loops of intestine, thus removing them from the circulating blood and tissue spaces. The chemical changes in the blood under these conditions, viz., decreased water, sodium and chloride contents and increased urea concentration, have been recognized for many years. Furthermore, clinicians have for a long time appreciated the therapeutic value of intravenous salt solution. The original hypotheses assumed that the condition was due to a toxemia and that the saline therapy acted as a detoxicant. Gamble's careful balance experiments finally proved that for pyloric obstruction, at least, no other factors than salt loss and dehydration were required to produce the entire disease picture. As in cholera and diabetic acidosis, striking clinical improvement follows the replacement of salt and water.

Underhill and others have studied the serious consequences of extensive burns and have demonstrated that the exudations into the burned area deplete the blood to such an extent that fatal shock may ensue. Animal experiments by Blalock indicate that it requires approximately the same total fluid loss whether by hemorrhage or as edema fluid in burns to cause fatal circulatory paralysis. More recently Harkins has studied the causes of death by freezing and has observed that there is a definite similarity to the effect of burns, viz., extensive loss of fluid from the circulating blood into the damaged tissues. Of course, I would not assert that there are no other factors contributing to the pathology of burns, but certainly there is none so well understood or so practical an aid to therapy as the fluid depletion.

The clinical syndrome of acute adrenal insufficiency as seen in Addison's disease bears a striking resemblance to that present in the other pathologic states which we have discussed. For example, weakness, prostration, rapid pulse, nausea and vomiting, fall in blood pressure, decrease in water content of the blood, increase in blood urea and decrease in the concentrations of chloride and bicarbonate are characteristic. Indeed, Addison in his original description commented on the similarity of the terminal stage to that seen in cholera. Loeb, in our clinic, has shown that in adrenal insufficiency in man, the sodium of the blood is markedly lowered, as Marine and Bauman and also Zwemer had shown in cats. Furthermore, he was able to show that the administration of sodium chloride alleviates to a striking degree the clinical manifestations just described. On the other hand, it is possible to precipitate an acute and alarming attack of adrenal insufficiency by the withdrawal of salt from the diet of patients who are in relatively good clinical condition as a result of salt therapy. It is truly remarkable to observe the difference that 15 Gm. of salt a day will effect in the health and well-being of such a patient. Loeb and I have demonstrated that the decrease in salt content of the blood in adrenalectomized dogs is due to an enormous increase in the excretion of sodium by the kidney. This loss of sodium is accompanied by an augmented but not parallel water output and results in the characteristic picture of dehydration, salt depletion and shock. It seems likely that the mechanism by which salt depletion and dehydration are produced in adrenal insufficiency is different from the disturbances resulting in shock in the clinical conditions described previously. On the basis of the evidence so far accumulated we are inclined to believe that the active principle of the adrenal cortex exerts a controlling influence upon sodium metabolism through the medium of the kidney, and that the breakdown of this regulatory mechanism results in an increased rate of sodium excretion. The regulatory effect of the adrenal cortex upon salt and water metabolism is not its sole function. This seems proved by the fact that adrenalectomized animals eventually succumb although the period of survival may be appreciably prolonged by salt administration.

Clinical descriptions of heat prostration suggest the shock syndrome and the observations made by the Harvard group at Boulder Dam support the suggestion that salt depletion and dehydration play a prominent rôle in this condition. Replacement therapy in the acute phase has been successful and preventive measures directed to the conservation of base have been valuable.

It has been shown experimentally that the rapid death in shock that occurs in so-called bile peritonitis is due in part at least to dehydration of the body by loss of fluid into the peritoneal cavity. This type of reaction illustrates clearly the fact, at times overlooked, that it is not necessary that the fluid and salt be lost from the body to the external environment in order to initiate the shock syndrome. A sufficient quantity of fluid statically mobilized in the tissues or body cavities will produce the same result because such fluid is no longer available to the circulating blood stream. Most modern workers in the field of traumatic shock feel that exudation into damaged tissues with its removal of large quantities of fluid at the cost of the blood volume is a potent force in causing this type of shock. There is ample experimental evidence to support this point of view.

It is apparent from the foregoing discussion that the loss of water and salt from the body may occur in a variety of ways and result from a number of different physiologic disturbances. Among these we have described: loss of base and water by diarrhea, vomiting or surgical drainage; loss of serum by exudation into burnt or frozen tissues; loss of salt in the urine by alterations in the carbohydrate metabolism; loss of salt as a response to acidosis; an excessive urinary excretion of sodium in adrenal insufficiency; and finally, unusual loss of salt and water by the skin following exposure to heat. There is one common denominator to be found in all of these disease conditions when they have reached an advanced state, viz., the shock syndrome.

The train of events by which dehydration and salt depletion ultimately lead to the classical syndrome of shock requires only brief mention. One of the most striking physiologic principles of the body is expressed in its tenacious effort to maintain the salt content of the blood serum and the interstitial fluids at a constant level in the face of adverse circumstances. When salt is lost for any reason, the body sacrifices its previous water stores to protect its sodium concentration. Conversely, the extensive loss of water is almost invariably associated with a considerable loss of salt. Hence, a severe drain of salt or water or both is reflected in the circulating blood, producing in time a decrease in blood volume so great that the state of shock ensues in much the same manner as in acute hemorrhage.

Let us next turn our attention to those situations in which the state of shock is brought about primarily by a rapid dilatation of the vascular bed rather than by contraction of the blood volume. Since the isolation of histamine by Sir Henry Dale in 1909, physiologists have recognized that large doses of this drug will cause a generalized capillary dilatation, with a drop in blood pressure and collapse. The closest clinical approach to this laboratory experiment is seen following the entry of venom into the body by the intravenous route. It was my privilege some years ago to observe the clinical effect of an intravenous rattlesnake bite. The patient, while extracting venom during his duties at the American Museum of Natural History, was bitten on the back of his hand. In spite of the immediate administration of antivenom serum the patient collapsed in twenty minutes and was brought to the Presbyterian Hospital. On admission he presented an extraordinary picture. His skin was cold and dusky red in color, he was semicomatose, his respirations were shallow, his pulse was rapid and almost imperceptible, his heart sounds were inaudible and his blood pressure was too low to be read. In spite of several saline infusions and a transfusion of 700 cc. his blood pressure, which was raised by these therapeutic procedures, soon fell to a critical level again. It was not until 7200 cc. of fluids had been given intravenously in the course of sixteen hours that his blood pressure remained normal. The frequently fatal outcome from an intravenous rattlesnake bite results from failure to recognize the fact that the therapeutic attack should be primarily directed against the state of shock. A recent study by Blair of the bite of the black widow spider brings to light many similarities between the effect of the spider toxin and rattlesnake venom on the peripheral vascular bed.

When Laennec, in 1826, described the weakness of the heart sounds in severe febrile conditions, he attributed this change to cardiac failure, a point of view which, unfortunately, still continues to dominate medical thought. This is true in spite of the fact that Romberg and Pässler as early as 1899 wrote upon the effect of bacterial products on the vasomotor apparatus of rabbits. These authors were able to show that a state of collapse could be induced by the intravenous injection of pneumococci or other organisms. Furthermore, they pointed out that intravenous salt solution was more effective in treating these animals than was subcutaneous ether, camphor, strychnine or cognac. Romberg at that time suggested the term "toxic shock" for this complication of infectious disease. It is hardly necessary to emphasize the similarity between Romberg's

"toxic shock" and the shock resulting from intravenous injections of histamine or snake venom.

A striking confirmation of the shock-producing capacity of the pneumococcus was afforded by a clinical experiment performed upon himself by a member of our staff a few years ago. This individual gave himself a large dose of pneumococcus vaccine, intravenously. There followed an immediate and alarming collapse during which his systolic blood pressure fell to 60 mm. of Hg. and continued below normal for three days. In conjunction with this he developed numerous petechial hemorrhages of the skin, which would lead one to infer that, in addition to dilatation of the capillaries, there had also been actual damage.

Doubtless you recall in your own experiences with severe infectious diseases, particularly pneumonia and typhoid fever, patients who have presented the picture of falling blood pressure, rapid pulse and collapse. In many instances this serious complication is a manifestation of shock, and probably results from an increase in the vascular bed due to capillary damage. Confusion of this state with cardiac failure will result in misdirected and possibly harmful therapy. The importance of this point of view was indicated by Theodore Janeway in the *New York State Medical Journal* for 1907, when he wrote on "Some Common Misconceptions in the Pathological Physiology of the Circulation and Their Practical Significance." He said, "We must in most cases abandon the idea of cardiac death at the height of acute infectious diseases such as pneumonia, typhoid fever and septic fever. . . . In place of heart failure we must write vasomotor failure." The discussion of this complication of infectious disease in many modern textbooks of medicine offers a discouraging contrast to Dr. Janeway's enlightened attitude.

There is still another mechanism involved in the production of shock through vasodilatation. I refer to the influence of the sympathetic nervous system. Surgeons recognize this influence in the production of primary traumatic shock and it is by this route that the well-known effects of fear and cold are exerted, although the concomitant vasoconstrictor effects via the adrenals make this a complicated physiological problem. In the field of internal medicine, we have come to realize that one of the immediate effects of extensive infarction of the myocardium is a clinical picture of falling blood pressure and collapse, which is identical with shock. It is hard to believe that such a rapidly developing syndrome could result from tissue damage unless it were brought about through a reflex nervous mechanism. Fishberg has made a careful study of this type of shock and his conclusions are of great interest to the cardiologist. He believes that the development of peripheral vascular failure in cardiac infarction is a fortunate physiologic disturbance because the return flow of blood to the heart is inhibited by the capillary stasis and the contraction in blood volume decreases the burden on the heart. Therapeutic measures to combat such a beneficial complication would naturally be contraindicated. However, other clinicians disagree with this point of view and feel that shock even under these circumstances should be actively treated.

Up to this point I have dwelt upon the occurrence of shock in a variety of clinical conditions, stressing in each instance the mechanism which dominates the picture. In some of these situations shock resulted primarily from a rapid decrease in circulating blood volume, whereas in others the chief factor was vasodilatation. It is only fair to state that this is a diagrammatic visualization of processes which like other biological phenomena are more complex than I have seemed to indicate. For example, even in histamine shock, where the primary disturbance is admittedly due to capillary dilatation, nevertheless capillary dilatation is inevitably accompanied by an increase in the size of the capillary pores which permits large quantities of blood serum to escape into the tissue spaces. Thus the factor of decreased circulating blood volume also plays a part. This effect may naturally be assumed to exist in the case of snake venom and bacterial toxins, or in any other condition in which generalized capillary dilatation is marked.

In diabetic acidosis accompanied by the shock syndrome, I have indicated the importance of dehydration and salt depletion. There are occasional patients in whom the state of shock persists after adequate replacement therapy and after the ketosis has disappeared. Certain experimental evidence suggests that there may be in these cases a factor of capillary poisoning which is responsible for the persistent recurrence of the shock syndrome. Thus, it has been shown that a variety of substances which have a chemical similarity to the ketone bodies, such as acetyl acetone and sodium acetate, will produce vasodilatation in animals.

I would like to digress, if it be digression, at this point and introduce briefly the subject of surgical shock. As an internist I am occasionally asked to pass on cardiac competency in relation to a projected operation. I am wholly in agreement with the work done at the Peter Bent Brigham Hospital which showed the surprisingly small effect operations had upon cardiac function. Much of the so-called cardiac and renal failure postoperative is due to shock. We have in this situation many forces leading to dehydration as well as the neurogenic factors producing peripheral vascular paralysis. At the risk of obvious triteness, I would point

out that salt solution is more often needed than digitalis. In these cases, as in any others where there is a real problem of differential diagnosis between primary cardiac failure and peripheral circulatory paralysis, observation of the venous pressure is a very helpful aid to discrimination. In shock it is usually within normal limits whereas in a cardiac state severe enough to be confused with shock it is usually elevated. As an exception, I must cite the occasional occurrence of very high venous pressures in pure shock due apparently to constriction of the veins, at least they are greatly contracted wherever visible. This finding is in distinct contrast to the venous distention seen in cardiac failure.

There has been much discussion of the participation of a toxin in the production of the clinical manifestations of intestinal obstruction and, indeed, substances with histamine-like action have been isolated from damaged loops of gut. To what extent these substances contribute to the total mechanism in producing shock is still problematical. I have pointed out the rôle of dehydration and salt loss in severe burns but here, too, it is possible that certain toxic substances are absorbed from the necrosing tissue and may act as capillary poisons. However, one inevitably returns to the fact that the toxins are hypothetical and as yet without therapeutic possibilities, whereas the other factors are well understood and easily treated.

The treatment of shock is more or less independent of its cause. Whether it be due to trauma, toxemia, hemorrhage or dehydration, the physiologic problem is the same, namely, a disparity between the circulating blood volume and the vascular bed. On one hand there is primarily a decreased blood volume from hemorrhage or fluid loss; on the other, an increased vascular bed resulting from capillary dilatation. The need for immediate measures to increase the circulating blood volume is common to all types.

I think it is reasonable to say that the longer the state of shock is permitted to exist, the more difficult it becomes to alleviate it and the higher is the mortality. Consequently, delay in initiating therapy is dangerous. As an emergency method, the intravenous injection of 50 cc. of 50 per cent glucose, which may be conveniently kept on hand, is of some temporary value, as it will draw fluid into the blood stream from the tissue spaces. Protracted use of glucose solutions is not only inadequate therapy but actually augments the state of shock by its tendency to cause dehydration. Glucose should be followed promptly by the intravenous injection of 1000 to 2000 cc. of normal saline solution. There is, among clinicians, a very general belief that the intravenous administration of fluids in large quantities tends to constitute a dangerous strain on the myocardium. This opinion is derived largely from experiments on animals not in a state of dehydration. Experiments in our clinic by Caughey have shown that a normal man can receive 1500 cc. of normal salt intravenously without altering his venous pressure. Dehydrated patients can accept many times this amount with no difficulty. If shock exists and there is no primary cardiac damage the response of the arterial blood pressure may be taken as an indication of therapeutic success. If there be any question as to the cardiac status a venous pressure manometer should be inserted into the infusion apparatus, and a rise in venous pressure used as an index of overload of the venous circulation. As I have just stated above, such overload is very rare even when several liters of fluids are necessary for satisfactory replacement.

Although a normal physiological solution of sodium chloride is usually an adequate therapeutic agent, it should be remembered that in many of the conditions that I have enumerated salt depletion is relatively more rapid than fluid loss. Under such circumstances it is wise to use higher concentrations of salt such as 1 to 2 per cent or even 5 per cent, until the restoration of base is complete.

The ideal treatment for advanced shock is a large blood transfusion, and every patient likely to develop shock should have his blood grouped early in the course of his disease. When salt solution has failed, transfusion may turn the tide. The theoretical basis for the fact that blood is more effective than salt solution lies in the assumption that it contains a relatively nondiffusible substance, i.e., serum protein, which makes the influence of added blood a more permanent one. We cannot accept this suggestion without reservation, for it has been clearly proved that in histamine shock whole serum leaves the blood stream; in other words, the capillaries become readily permeable to protein. However, there is no denying the greater therapeutic value of blood over all other fluids. In an attempt to find a substitute for blood, other colloidal substances have been tried. The most prominent of these is acacia. During the World War, this solution was tried but given up in this country because of the severe reactions which frequently followed its administration. In the past few years, methods of preparation of acacia solutions have been improved, and enthusiastic reports of its action have appeared.

All therapy in severe shock should be intravenous rather than subcutaneous or intramuscular. The rapidity of response is much greater when the intravenous route of administration is employed, because the poor peripheral circulation tends to slow subcutaneous absorption tremendously. This applies to hypodermic medication as well as to fluid administration.

The use of vasoconstricting drugs such as adrenalin is not helpful and may, in fact, be harmful. From a physiologic standpoint they are contraindicated because the blood vessels which they affect are already constricted to the disadvantage of the capillary circulation, as has been shown by studies of both the skin and visceral arterioles. Furthermore, experimental work has shown that continuous injection of adrenalin in quantities that are equivalent to the amounts produced by sympathetic stimulation can cause a 14 per cent decrease in blood volume during a two-hour period. Hence adrenalin exaggerates the state of shock, even though it temporarily increases arterial pressure.

In conclusion I would like to express my pleasure in being offered an opportunity to review this subject before a group interested primarily in cardiac problems, for it is the cardiac consultant who most often has to assume the responsibility for differentiating peripheral circulatory failure and the condition with which it is so commonly confused, viz., cardiac insufficiency. The most satisfactory point of orientation lies in the recognition of the constantly recurring basic physiological pattern, resulting from a disparity between the circulating blood volume and the vascular bed. Although I am aware that much of the ground that I have covered is familiar to you, the lack of emphasis upon this important complication of disease in the most modern textbooks justified, I felt, my making the summary as broad as possible.

THE ECONOMIC AND SOCIAL ASPECTS OF SOCIALIZED MEDICINE*

BY IAGO GALDSTON, M.D.†

THE American medical profession is at this time confronted by a social movement which threatens to disrupt the profession's individualist relation to the public. This movement has for its objective the socialization of medicine, and while there is no agreement among its proponents as to the specific form which the socialization of medicine should take, we may assume with certainty that what is contemplated differs radically from the prevailing quid pro quo individual relationship between doctor and patient.

In an objective analysis of the movement for socialized medicine, we must begin by seeking for its origin among social and economic factors. "Why", we must ask, "is there such a strong movement for the socialization of medicine?" The answer in part is, that the movement has arisen in response to needs of an economic and social nature. There is undeniably a great need among our people for additional and more extensive medical services of a curative and prophylactic nature. There is a mountain of testimony available to the effect that this nation suffers much economic loss as well as much misery by the prevalence of preventable and inadequately treated illness.

Together with this realization there is prevalent a strong conviction that by a more effective utilization of the available resources of medical science, the average lifetime could be extended, much illness could be avoided, and much of economic loss saved. This dilemmatic situation raises the pertinent question: "Why does not more of our citizenry receive adequate medical care?"

Among the many answers given to this query, the outstanding one is, that present-day medical service is too costly. "Too costly" may have one of two meanings, either that the return received for the expenditure is of too small a value, or else that the commodity is beyond the means of the potential purchaser.

It is in this second meaning that medicine is complained of as being too costly. The average American family cannot afford to purchase for itself adequate medical care. Pushing our inquiry farther, we may ask: "Why cannot the average American family afford to purchase adequate medical care?" The answer to this query is to be found in the records of the distribution of our national wealth and in the distribution of our national income. According to W. I. King*, sixty-five per cent of the people in the United States own but fifteen per cent of the wealth of the country, the maximum average wealth in this group being less than $3,500. The remaining thirty-five per cent of the people comprising the middle and rich classes own eighty-five per cent of the national wealth, and in this group two per cent of the people own forty per cent of the total wealth of the country.

Perhaps even more significant, from our viewpoint, is the distribution of our national annual income. According to the National Bureau of Economic Research‡, 66.7 per cent of our income-receiving individuals have an annual income of between $500 and $1,500. An additional 13.9 per cent have annual incomes ranging between $1,500 and $2,000. In other

*The views expressed herein are those of the author alone and do not in any way represent those of any institution with which he is or has been connected.

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†Galdston, Iago—Secretary, Medical Information Bureau, New York Academy of Medicine. For record and address of author see "This Week's Issue," page 884.

*Wealth Distribution in Continental United States, W. I. King, *Journal of American Statistical Association*, June, 1927.

‡Income in the United States, National Bureau of Economic Research, pp. 136-7.

words, 88.8 per cent of the people receive but 58.7 per cent of the total income. In contrast we find six per cent of our people receiving roughly forty per cent of the annual national income.

These figures reveal why adequate medical care is too costly for the vast majority of people. These figures reveal also, though they do not tell the entire story, why there is so much preventable illness and avoidable loss of life. These figures explain why, despite the wealth of available medical knowledge and the richness of our scientific resources, so many of our people are without their benefits.

We are thus brought to the realization that the movement for the socialization of medicine arises out of the complexities and imbalances of the prevailing economic system.

Fundamentally, too, we must realize that the socialization of medicine is an attempt to secure adequate medical service for the average man and his family without going through any radical change in the distribution of national income. The only way in which this might be accomplished is by compelling the physician to render more service for proportionately less remuneration. This must be plainly evident to anyone who seriously contemplates the problem. As long as the average income of the average family remains what it is today, only a small portion of that income will be available for the purchase of medical service. No matter how this portion is collected, whether by a system of voluntary taxation, by insurance, or by any other method, it will still remain a small per unit volume. By the expenditure of this collective sum only so much medical service is secured today, and by common agreement, this amount of medical service is insufficient. If more medical service is to be secured for our people for the same collective sum, this can be accomplished only by lowering the price of medical service.

The problem which the medical profession faces today and which society as a whole faces with regard to medicine, is part and parcel of the larger economic and social problem which enmeshes all of mankind. Its origin is identical with that of the economic disorganization which has imposed a pall of depression upon the entire civilized world. There is little hope that any effective solution of the medical problem will be achieved until the underlying economic factors are set aright. Nor, for that matter of fact, is there any hope that we may effectively escape the periodic disruption of our economic existence until a basic readjustment in the distribution of the wealth produced is accomplished.

However, as a profession we are rooted in individualism; but it is an individualism with a large social consciousness. What other communal group can match us in our free gifts to society, of our toil, of our thought and of our genius? Where other men have exacted the utmost of their so-called due, for their discoveries, their inventions and their contributions, our ethics and traditions have taught us to make freely available to all mankind all of our finds. James Watt patented his steam engine in the same age that Jenner discovered smallpox vaccination. Edison invented and patented the incandescent lamp about the time that Von Behring discovered diphtheria antitoxin. Banting gave the world insulin in the same decade that the DeForest valve made the radio one of the major modern industries. Let history judge which were the greater gifts to mankind, and let it bear witness to the enlightened individualism of the medical man.

We have made a necessity out of a virtue. Charity is not a sabbatical practice with our profession, but our everyday performance, so ingrained in our relations to our fellow men that they largely demand it now as their due.

There is more to this problem than appears on the surface. Thus, it is not pertinent to inquire: Why do our people require so much medical care? We who are physicians know the effects of economic status upon physical well-being. Health is affected by food, clothing, shelter, work, leisure, education and recreation. Is it not possible that the reason why our people require so much medical attention is not because they cannot afford adequate medical service, but rather because they cannot afford adequate food, clothing and shelter, because some are worked too long and too severely, while others are left to disintegrate in idleness because their education is inadequate and too poor, etc.?

It is pertinent to observe that when a man is sick, he has the powerful vis medicatrix naturae fending for him. He may get well without the aid of his doctor, sometimes even despite his doctor. But who fends for man in want of food, in want of shelter, in want of clothing?

Germany has had socialized medicine for fifty years, but the average expectation of life at birth in Germany is about ten years less than in our own country.* Evidently the socialization of medicine works no miracles.

Hence we come to a point in our consideration where we acknowledge the need for improvement in the medical care of our people, but reject the proposal to socialize, that is, to industrialize medicine, because it offers no promise to remedy the existing defect, because it would involve the disorganization of the profession of medicine, with the loss of so much that is best in it.

When the proponents of socialized medicine tire of harping on the alleged high costs of present-day medical care, they turn historians.

*Health and Wealth, Louis I. Dublin, pp. 17-18.

With a majestic sweep, they encompass the story of the ages and they distill the "unquestionable lesson from history." They give utterance to such generalizations as "Everywhere and always, the physician has been a product of his times, and the conditions under which he has practiced have invariably reflected the customs of the period."

The crux of the entire matter appears best expressed in the epitomic statement, uttered by those favoring the socialization of medicine as if it were axiomatic, to the effect that: "The physician's position in society is never determined by the physician himself but by the society he is serving."

This pseudo-historical dictum needs careful scrutiny. To begin with, the question might be asked: In society what professional group ever succeeds in determining outright and without opposition its own status in the communal economy? Further, the protagonists of socialized medicine use the term society in an animistic sense, as if society were a corporate thing, possessed of a unique intelligence which understands clearly the relationship of the different groups incorporated within it and is capable of assigning to each its proper rôle and position. This animistic concept of society is medieval in character and was discarded more than eighty years ago. *The position of medicine in this given society will be determined by whatever pressure those groups can bring to bear upon medicine in whose interest it is to purchase the services of the medical profession at the cheapest and the lowest possible rate and also by the amount of resistance that will be shown by the medical profession to such exploitation.*

But disposing of this pseudo-dictum is not enough for our purpose. We must pursue the subject farther. There was for those that propounded it an end which the dictum served. Let us follow to that end. The almost syllogistic reasoning ran somewhat as follows: "Medicine never determines its position in society; on the contrary, society does that for medicine. Our society is determined to socialize medicine; hence, it is foolish for medicine to oppose the inevitable." In the words of Sigerist, "We can oppose the development, we can retard it, but we will be unable to stop it", or, as it is paraphrased by others, "In any final sense, the economic and professional needs of modern medicine call for group payment by the public, group practice by the profession and a conjunction of the two."

We must ask here: Who is it that defines the "final sense" and who is it that determines the economic and social needs of modern medicine? Since there is no corporate state, no articulate society, who is it that has been pretending to the rôle?

Undeniably the social trend is toward socialization. But "trends" have no transcending warrant to exempt them from critical scrutiny. Trends can be wrong, too.

In this connection it is interesting to note that every age has its shibboleth, has its linguistic gilded calf to which the deluded bow and pay homage. The shibboleth of our age is socialism and socialization. So powerful and so narcotizing is this term, that even the most arrogant of dictators make use of it. Fascism in Italy justifies itself in the name of socialization and Naziism in Germany even goes so far as to call itself National Socialism.

And in truth, the word social has a fatal enchantment, for to the unthinking, whosoever is against socialization appears per force, to be for greedy and thoughtless selfishness. Few construe the opposite of socialization to be individualization. Nor is it commonly recognized that one may be for the socialization of certain phases and functions of human existence and endeavor, and yet be for unconditional individualization in other respects. Total and unconditional reactions are usually associated with juvenility and inexperience. Few love or hate so completely as the child or childish adult, and in the widespread embrace of the idea of socialization which, like the conversion manias that swept through Europe in the early centuries, have now involved so many of our vocal thinkers, there is more hysteria than religion, more quest for refuge from perplexity than understanding. Because individualism and laissez-faire have brought in their train so much suffering, therefore let us destroy individualism, let us socialize! All of mankind's history has been like the swing of the pendulum, a progression from extreme to extreme, and never has the golden mean of the Greek philosophers guided us in our affairs. No man can be insensible to the sufferings for which modern capitalist society is responsible; no man can be so blind as not to see the greatness achieved by the free and unfettered enterprise of the individual entrepreneurs. Now, as in all times, in Athens, in Republican Rome, in the free cities of Italy, the greatest ages have been those of individual freedom.

Let me not be understood as being opposed to social endeavors, not even to much of that which is properly called socialism. Many phases of human existence can best be served by social endeavor. Our own profession, that of medicine, though practiced by individuals and in an individualistic manner, is in its studies, in its research, in the inter-relationship of practitioner to practitioner, and the total relationship of the profession to the people, a most highly social profession. My protest against the shibboleth of socialization is that it is offered

as a panacea, as an ultimate and final cure-all, as an unfailing remedy. Of course, it cannot be and is none of these.

The proponents of the socialization of medicine have no adequate understanding of the psychologic nature of man. Their reasoning follows the guidance of a naïve materialism. Because the chain belt system of mass production has given us cheap and effective automobiles and radios, sewing machines, farm tools and mail order house products, therefore, they reason, a similar system of mass production and distribution can be as effectively applied to medicine. But they count without the nature of the beast. Man is a gregarious animal. On the least impulse he will flock with his fellow kind. He loves the group and feels strong in the company of his own. But for that very reason he is as strongly in need of periodic affirmations of his singularity, of his uniqueness, of his individuality. Man can toil in common, but save for the ceremonial banquet he prefers to eat alone, or with those very few who are near to him. Man can, to an appreciable degree, sink his ego in the complex organism of the factory, shop, office or community, but only at his peril can he afford to abdicate his birthright of individuality as a lover, father or elder. *One might with warrant generalize that man is a social, communal animal as a producer, but an individualist as a consumer.* And in relation to the physician, the patient is ever a consumer.

There is in this struggle between those who urge the socialization of medicine and those who oppose it a deeper cleavage than appears from even a careful pursuit of their arguments.

To me it seems that in this difference between the two groups, there is reflected two fundamentally divergent philosophies, as far apart as those two that motivated Athens and Sparta. It is not merely a question of the socialization of medicine but more it is a question as to which of two lines of pursuit offers economic and social salvation.

Shall it be the state, the patriarchal institution upon whose lap we shall lay the full destinies of the individual, to whom we will look for the regulation of our living manner, and the provision of our multiform needs, or shall it be the individual, grown to the stature of a civilized and social being who will as a sovereign carve his own destinies, in the company of, with the help of, but at the cost of none of his fellow men?

In other words, shall men rule themselves in all respects of life and be truly democratic or must we turn back to dictatorships?

There is, in the practice of medicine, a saving grace which as in the case of the isolated tiller of the soil defends the practitioner against becoming brutified. That grace in both instances is intimate contact with life. The farmer is an intimate witness of life in the things that grow about him, the physician in the human beings that claim his help. The rest of the world deals with things, with goods and money, with machines and figures and books. These two, the physician and the farmer, shall we say, "see life in the raw".

It is perhaps for this reason that both agriculturists and physicians are conservative. They hold to their values and are not easily persuaded to exchange them for new script. So even without a knowledge of the lessons taught by history, the physician is an individualist and a democrat, and the profession is well represented among those who fought for the rights of the individual from Servetus to the medical signatory of our Declaration of Independence.

The issue of democracy has been raised once again, and there are some who, like the benighted Athenians, are issuing a call to Phillip. The medical profession in its small segment of this matter and in a stammer indeed, is sounding the Phillipic warnings of Demosthenes. We distrust the tyrant, even when he comes garbed in the cloak of socialization. We claim for ourselves and for mankind the birthright of suffrage. We say the other way, the way of the Caesars leads to Caligula and Nero, to the dominance of the barbarians, to the yoke of the Roman Empire. We remember how mankind fought for the Magna Charta, for the Reformation, against the Louis of France and the Georges of England. Our faith then, is like that of Pericles with the Demos. This Demos, our philanthropist, however, does not understand, nor does he love it truly.* How simple is his reasoning! Cavalierly he lumps together all social insurance schemes, unemployment, old age, disability and sickness, never thinking that the first three are merely matters of compulsory savings, while sickness insurance involves the uprooting of an entire profession and a shifting of the stream bed of motivation that has for so long been fixed between patient and physician. Furthermore, it means bureaucracy, that cankerous parasite that always comes into being when human relations become indirect, complicated, and achievable only through intermediaries. *What is there in our history, in our experience, that warrants faith in the successful socialization of medicine?* We are a nation of people honest as individuals and often cynically corrupt in the mass. Our history is taught badly, or else we would better appreciate the veniality of our collective people, for this is the land of graft, collusion and dishonesty. How short is our memory? Carpetbaggers, poisoned beef for our Spanish-American War soldiers, Tweed, Teapot Dome, the banks; nay, Gustavus Meyer's history of great American fortunes, the autobiography of Lincoln Steffens, and Beard's American History,

*Vide Dialogues in Limbo—Santayana.

should dissuade everyone from faith in the governmental, mass or mob. To this, add our own experiences with workmen's compensation and with communal and government hospitals and the picture is complete.

Lest I be charged with misanthropy, let me hasten to add that no man is to blame who fails, when what is required of him is beyond all limits of his capacity to achieve. Man can love his kin, respect his neighbor and share in the suffrage of his town. Beyond these only the exceptional man can reach. For this reason the Government's post office pen is no one's pen and tax money is a common source of political patronage.

Despite this, there is no doubt but that the agitation for socialization is very intensive. An indication of the economic and social confusion that prevails is to be witnessed in the fact that the socialization of medicine is sponsored by two diametrically opposed economic groups. Socialized medicine is endorsed by the revolutionary groups, and it is sponsored as well by the reactionary groups. The one sees in the socialization of medicine the cutting wedge of their longed-for coöperative commonwealth. The stand-patters see in the socialization of medicine a prop for the present economic system. History, I suspect, will prove both in error.

At this point it is important that we turn the limelight of criticism upon ourselves. Because we are so bitter in our criticism of the proposed schemes for the socialization of medicine, let it not be construed, therefore, that we are without fault.

The medical profession has unquestionably shared in the predatory ideology and practices of present-day society. We may talk of the nobility of our profession and may cite, as I have, numerous individual examples of professional generosity and unselfishness. In fact, it can be argued that few professions have so many such illustrious examples in their membership as does medicine. But the public judges us, and rightly so, not by our illustrious examples, but by common denominators, and these have been confessedly none too good, at least when measured by our pretenses and by our numerous privileges, which impose exceptional obligations upon us. This has been as true of the upper group as of the lower.

Need I at this time remind you that medical education for the undergraduate was until some thirty years ago largely a commercial endeavor, carried on for the profit of the entrepreneurs? Need I point out to you that the solidity and unity of medicine, culturally and otherwise, is disrupted by the existence of numerous small cliques, exclusive societies and the like? We may indulge in the orator's exaggeration and speak of our noble healing cult and of our oath of Hippocrates, and yet, a minority of fortunate physicians have for many years held on to their hospital, dispensary and other positions, concerning themselves but little with the very large number of physicians who are without such opportunities for continued study and training.

It is well to talk of spiritual enslavement and of the submergence of the individual ego, but when one is economically enslaved, when one does not know how to make ends meet, one's so-called birthright appears like a small price to pay for a mess of pottage wherewith to dull the gnawing pains of hunger. Undeniably, even among our own ranks there are many physicians who would be eager to accept the socialization of medicine precisely because therein is the promise of more than they can get out of the "dog-eat-dog" society of today. This circumstance is very unfortunate. One cannot and one must not shut one's eyes to the suffering and deprivation which is about us. We, however, who are physicians, must not "jump at" a remedy which threatens to be worse than the disease.

Whether the agitation for the socialization of medicine will prevail I am unable to say. My guess, which is no better than the next man's, is that the cards are stacked against us. However, be that as it may, if I read history aright, there is a "triumph of the defeated" no less great than the triumph of the triumphant. The effectiveness of opposition is a chapter in history which few appreciate, and the virtue of opposition as a social instrument I recommend for consideration. The Greeks were defeated by the Romans, but the satires of the later-day Roman poets (Juvenal) reveal that the Greek, rather than the Roman, prevailed. Rome was overrun by hordes of barbarians, but the invaders were Romanized. Christianity triumphed over paganism, but some of our customs are witnesses of the triumph of paganism in its defeat.

Our opposition to the socialization of medicine must therefore be persistent. We are bound to triumph even if we are defeated. Our opposition, however, must be discriminatory rather than total, and it is our eternal obligation to deflate the pomposities of the promoters of pet schemes to show up their shallow thinking and their ignorance, even as I hope I've been able to do.

MEMORIES OF A GREAT PHYSICIAN, DR. FREDERICK C. SHATTUCK, OF BOSTON

BY JOHN B. HAWES, 2ND, M.D.*

"HALF life is memory; the other half anticipation", says Yeats-Brown. Having reached an age when the years crowded with memories far exceed those I may rightfully anticipate, I am hereby jotting down certain very precious recollections of my early medical life and especially those years when it was my privilege to be the assistant to the late Dr. Frederick C. Shattuck of Boston. Dr. Shattuck, then at the height of his career, was professor of clinical medicine at the Harvard Medical School and chief of the Medical Staff at the Massachusetts General Hospital. He was a "doctor of the old school" as was the original of that name so vividly described by Ian MacLaren and who for forty long years gave loving care to the dwellers in Glen Drumtochty. Like him, Dr. Shattuck practiced medicine as an art, making a room brighter by his presence and trials and tragedies easier to bear for those who must bear them. The incidents I am about to describe took place during my first twenty years of practice when I, as a young practitioner in Boston, was intimately associated with him first as student and later as his assistant.

Dr. Shattuck was a picturesque figure, handsome and what one might call debonair. His face, always attractive, lit up on smiling so that it radiated cheer. Strikingly dressed, his waistcoats, invariably of some brilliant plaid, have gone down in song and story. A flower in his buttonhole and a cigarette that hung in some miraculous fashion from the corner of his mouth completed a picture that one always looked upon with pleasure.

It was a morning in October just thirty years ago when the shininess of a new brass shingle outside the door of my office on Marlborough Street had had no chance to become dimmed that I was called to the 'phone and heard Dr. Shattuck's gentle voice asking me to drop down to see him for a minute. Much impressed and filled with curiosity I started out walking—we had no automobiles in those days and I did not consider my bicycle sufficiently dignified for the purpose. His office was small but like the man sunny, warm and cheerful with a fire burning on the hearth as it always did for at least nine months of the year regardless of weather. He was sitting in his well-worn leather armchair smoking as usual. "Come in", he said. "Sit down and be comfortable." I wondered what was coming next. "Hawes", with that quizzical smile I came to know so well, "I'm getting very old and Smith (referring to his then assistant) is getting very busy. I wonder would you be willing to assist me in some of my work?"

Would I be willing to assist Dr. Shattuck? There was no more enviable position in Boston for a young medical man than this and right well I knew it. I do not remember leaving his office but I do recall walking up Marlborough Street on air. It was a wonder that I was not pulled in by the police! Then followed many years of hard but intensely interesting work. The practice of medicine in Boston and the life of the young physician at that time was a very different thing from what it is now. I remember very distinctly consulting two men whom I looked upon as representing all that is finest in medicine as to what I should do and where I should open my office. Each gave me the same advice. "John", they said, "You must be on the staff of the Massachusetts General Hospital and have an office on either Marlborough or Beacon Street. These are the first requirements of a gentleman who wishes to practice medicine in Boston." How often have I thought of their advice with some amusement and yet with much respect. Incidentally, I followed their suggestions!

Getting started on a medical career in Boston was no easy matter for a young doctor with no money. Saturday afternoons, Sundays and holidays were religiously spent at the office hoping for a telephone call or that some medical crumb would fall from the plate of those older and well-established physicians whose names and reputations meant so much to us. How clearly incidents of those days stand out in my mind! A beautiful Saturday afternoon in my office to which the sun never penetrated, longing for the golf links or for a row on the Charles, I was reading with some difficulty a German medical magazine when the telephone rang and I heard Dr. Shattuck's voice: "Hawes, do you care to make a three-dollar visit in South Boston?" How glad I was to break away from improving my mind! On my faithful bicycle with my doctor's bag hanging on the handlebars across the city I went to earn and well earn my precious three dollars. One of my very first patients was a charming young girl who thought she had something wrong with her chest. Being a thorough and conscientious young man I told her to strip to the waist so that I could examine her and then modestly left the room. A few minutes later I returned to find that she had misunderstood my directions

*Hawes, John B., 2nd—President, Boston Tuberculosis Association. For record and address of author see "This Week's Issue," page 884.

and had removed her clothing from the waist down! Blushes on the part of each of us.

Again one beautiful Sunday morning I was walking down Beacon Street with an older physician, a splendid Irishman, loved by all of us and with a very large practice in the city. As we strolled along I was deeply impressed by the number of people and particularly the pretty girls to whom he doffed his hat and bowed. "My word, Doctor", said I, "What a wonderful practice you must have and what a group of lovely girls you are acquainted with?" "Hush, my boy," was his reply with a wonderful Irish twinkle in his eye. "Johnny, my lad, I don't know a one of them but don't you think a pretty face deserves a bow?" Joyous memories!

Another time a classmate and dear friend of mine who lost his life in Flanders—Dr. George P. Howe—was taking his daily exercise in a wherry on the Charles River. This was before the Esplanade was built and a dam controlled the tides so that rowing at low tide was a bit precarious and one had to watch the channel pretty carefully or else pay the penalty. My friend for some reason always known as "Peter", nearsighted to a great degree, had strayed from the straight and narrow path and found himself stranded on the mud flats where he had to get out and walk. The shells cut his feet and he was in anything but a good humor when a short way off a fellow oarsmen resting in deeper water called out and asked if he could help. To which Peter very logically but not tactfully replied: "I don't see what in hell you can do anyway." He was embarrassed and chagrined when he drew nearer and found out that his would-be Good Samaritan was one of his professors and instructors at the Medical School—Dr. Elbridge Cutler.

Dr. Cutler was a splendid example of the general practitioner of those times. Once he left his practice in my care while he attended a certain medical meeting. It was June so that most of his patients had moved out of town to their summer homes at the North or the South Shore. "Dr. Hawes", said he, "I am leaving five or six patients in your care. They are all well along in years and I hope that you will make no radical changes in the treatment I have laid out for them. You see, all that I am doing is to escort them pleasantly to the grave." This remark made a deep impression on me for I was young and believed in a somewhat militant and aggressive form of medicine. As I grow older I realize that more and more the task of us doctors in many cases is to escort our patients as pleasantly as possible to the grave. And I certainly made no changes in those that Dr. Cutler left under my care.

Always a gregarious fellowship, we young doctors were wont to foregather at mealtimes, there to forget for the time the dullness and trials of a medical existence. For a good many years there had been at 89 Charles Street a favorite eating place known as the P. O. T. Club which meant "Pay or Travel." One of us, I for one year, well earned the dollar a week deducted from our board by collecting it from the others. We learned from experience how to carve efficiently if not elegantly. Comments on any false move or failure to find the joint were loud and much to the point while vociferous demands for second helpings before the unlucky one elected to preside over the roast had had a chance to help himself stimulated speed and accuracy. A bowl containing marbles marked from one to twenty-five was passed around as a regular ceremony before dinner, each man drawing a marble. If he drew a low number his beer cost him accordingly while loud indeed were the lamentations of those getting the high numbers. The drawings completed and the money collected, one of us, usually my husky friend, Peter, went around to the corner grocer and returned with the ease of beer. Such eating clubs came and went but each left behind delightful memories.

But it was with Dr. Shattuck that most of my work was centered and it is of him that my memories are clearest and most cherished. At that time he was constantly being called in consultation to see patients all over New England while in addition during his term of service he made daily visits at the hospital and conducted clinics twice a week for third- and fourth-year medical students. These it was my job to plan and arrange for. There was an immense amount of laboratory work including specimens of all kinds to be examined, reported on and mailed each night before going to bed. Many evenings, tired out from a hard day's work or an equally strenuous party—for doctors do relax at times, I came home only to find a half-dozen boxes and bottles awaiting me which meant weary hours in the laboratory, painfully slow typing, sometimes half asleep, until finally the clank of a letter-box cover told me that I might go to bed. It was hard but interesting work.

The clinics I rejoiced in. Out of his vast experience there was always some new diagnostic point or valuable therapeutic procedure to be gleaned. On these mornings, Henry, Dr. Shattuck's coachman and later chauffeur, a familiar figure to all of us in the Back Bay, would drive the victoria and two sleek bays to my door and for the short distance to the doctor's office (and I always wished it were longer) I was quite the King, leaning back nonchalantly in the middle of the seat with a priceless sable robe thrown carelessly over my knees, hoping that some of my friends would see me.

I well remember one day when on reaching the hospital Dr. Shattuck said to me, "Hawes, go to the Drug Department and bring me a five-grain capsule of quinine. I have a cold." This I did and having done so ventured to ask why he took the quinine and what he thought it would do for his cold. I had imagined that the days of quinine, except for malaria, were past. An amused look came over his face as he replied, "I haven't the slightest idea what quinine will do for my cold but I know damn well it will make me more comfortable!" There was much sound common sense in what he said, particularly in those days of therapeutic nihilism.

Dr. Shattuck was eminently an honest man not only in his medicine but in every other way. He hated subterfuge and short cuts; he was firm in his belief that no special tests or signs could take the place of good thorough work that covered every detail followed by a serious consideration of all the facts in the case. In this way alone was it possible to arrive at a correct diagnosis. John Buchan expressed his feelings very well when one of his characters said, "The peril is that . . . men may be tempted to seek short cuts and the good God does not permit of short cuts in this life of ours." At one clinic the subject of exophthalmic goitre, now called hyperthyroidism, was under discussion. Now in every medical school class there are always at least one or two so-called "bright boys". They occupy front seats, listen intently and appear to be and perhaps are deeply interested. They are always ready with some question they believe to be pertinent and resemble the Bandarlog in the Jungle Stories in their desire to attract attention. They constitute a harmless but not a particularly admirable group. This is common knowledge to any doctor. On this occasion while the salient diagnostic points of the case were being demonstrated one of these students leaned forward and asked, "Dr. Shattuck, is not 'exophthalmos' a pathognomonic sign of this condition?" This was rather in the nature of a red rag to a bull. I waited breathlessly for his answer. Looking at him pityingly, Dr. Shattuck replied, "Young man, Christ said, 'An evil and adulterous generation seeketh after a sign and there shall no sign be given to it.' " The student collapsed and was heard from no more.

Dr. Shattuck believed in speaking the truth but not so as to hurt the patient if it were humanly possible to avoid so doing, as shown by the following incident which likewise shows why he was so loved by all who came in contact with him. This occurred when I, as his senior house officer, was escorting him through the hospital on his daily visit. On entering one of the wards he pointed to a very pale and evidently extremely sick woman in a nearby bed and asked, "Hawes, what is the matter with that patient?" Now the position of senior house officer is a very exalted one and the individual occupying it takes great pride in any rare or unusual case that he is able to demonstrate to his chief. He has not yet learned his lesson. Accordingly, I replied, loud enough for all to hear, "That, sir, is a case of pernicious anemia." A pained look came across his face. "Come here," and he led me across the ward out of hearing. "Primary anemia, please, Hawes, 'pernicious' has a horrid sound to the patient!" I have never forgotten that incident and then and there resolved that for the rest of my life I would deal with human beings and not with "cases" no matter how interesting they might be.

Another time I accompanied him to one of the surgical wards where his opinion was requested concerning a man who had been doing very badly after his operation. He read the history, studied the chart and very briefly looked at the patient who was evidently dying. Every effort in the way of injections of salt solution under the skin and other equally painful procedures as was customary in those days was being made to help him although the case must have been hopeless from the start. I handed him the consultation slip and again looking gravely at the sick man he wrote briefly on it and gave it back to me. "I should put no obstacle in the way of his peaceful departure," is what I read.

Dr. Shattuck was always intensely interested in the health and welfare of those associated or working with him. Various times and indeed once in my own case he prescribed vacations for his house officers when even to think of such a thing was the rankest kind of heresy. Rules and regulations were all very well but they held no fear for him and at times were meant to be broken. One of the important duties of an assistant such as I, was to "camp" on certain very sick patients who could afford it and for whom it was felt that the constant attendance of a young doctor on the case in addition to the nurse might be helpful. For three or four days I had been on duty at the bedside of a very prominent man, an intimate and dear friend of my chief, who was dying of pneumonia. It had been a constant but a losing battle trying in vain by subcutaneous doses of morphia, digitalis or strychnia to carry him past the crisis. I had thrown myself into this case most intensely and had just about reached the breaking point myself when Dr. Shattuck entered the room carrying a bundle done up in a newspaper. Looking at the patient and then at me, he beckoned toward the bathroom, a favorite consultation place for all doctors. Closing the door softly, he carefully unwrapped his package. "This, Hawes, is a bottle of Pol Roger, a favorite brand of your patient here. I think it will do him good."

Lovingly he undid the wire, uncorked the bottle and without saying a word took the bathroom glass, filled it to the brim with the champagne and handed it to me. "Drink that," he said. Decidedly taken aback I murmured, "No, thank you, sir, not while I'm on duty here." At once he replied, "Hawes, you're working for me and I'm paying your salary. You'll be of no use to me if you break down. Do as I say." And I did. Other camping incidents had their lighter side especially when the patient got well. Once I spent five days and nights in the cellar of a famous Boston banking concern where one of the firm had been taken suddenly sick and could not be moved. In wandering around at night, for there was not much to do, I found a cupboard containing a set of massive silver-mounted hair brushes for each partner with the owner's name engraved—and very awe-inspiring names they were! I amused myself each morning using in turn the brushes of this or that well-known financier until finally my patient was taken home through an opening they made in the wall so that he could be carried out on the level! I learned a great deal about high finance while on duty on this case. Again, late one night when in evening clothes at a party of my own, Dr. Shattuck sent me to a club in Boston where a Christmas celebration was being held at which a certain very delightful but somewhat frail old gentleman had collapsed, the cause of which I saw no reason to inquire. A holiday play was being given by the members and directly back of the stage was the bedroom where I found my patient sound asleep. There was high festival held that night with song and laughter accompanied by the popping of what seemed to me innumerable corks, all of which grew louder and louder, finally reaching a magnificent climax with voices not quite so clear and songs less steady, after which gradually the noise died down, leaving only a clatter of dishes as waiters cleared away and then silence. I had been very envious of those who had been having such a good time in front and quite bored with my somnolent patient and was glad when morning came when I took him to his home, both of us still in evening clothes.

Another time Dr. Shattuck gave me the somewhat unenviable job of taking a well-known citizen with marked suicidal tendencies by train to a certain institution an hour's ride or so away from Boston. The parlor-car seats were all taken so we sat down together in a crowded day coach. I did not enjoy the trip. Once he tried to escape from me and to jump off the train and again in a very loud whisper he asked me, "Dr. Hawes, do you really think I'm going insane?" At this a nice old lady seated directly in front of us got up hastily and scuttled into another car. I was quite relieved when I finally deposited him at the door of the institution to which he was going. Not a pleasant task but good training.

Dr. Shattuck's clinics and lectures were a delight to listen to. For the latest advances in science and the laboratory side of whatever subject he took up he depended on his assistant but the clinical aspect of the case was presented as none but a master could do it, based on years of thorough and thoughtful observation at the bedside of sick patients. A week or so before a given clinic on heart disease, for instance, the telephone would ring and I would hear his distinctive voice saying, "Hawes, I'm talking to the fourth year men on hearts next week. Look up all the latest work on the subject and let me have it, will you?" And up to the Medical Library I would go and wade through all the latest literature on the subject in English, French, German and sometimes Italian until I had collected for him what I knew he needed. Then it all had to be boiled down, typed out and handed in to him. When the time for the clinic came he would use this material as he saw fit but would invariably make it subservient to the clinical and human side of the problem. The comfort of the patient, whether sitting up in a wheel chair or lying in bed, was always his first consideration. No patient ever went back to the ward utterly exhausted and tired out as I have seen happen various times at clinics elsewhere. There was no perfunctory dealing with cold scientific facts. Always in his teaching he dealt with human beings who happened to have this or that disease and never with *cases*.

After the clinic came the hospital visit which was quite a ceremonial affair. Three or four house officers, senior, junior, "pup" and sometimes the externe, the head nurse, often some visiting physicians and always his assistant accompanied the doctor on his rounds, and—I nearly forgot—most important of all, there was Hans, a very elongated and rather pink dachshund, whom the patients loved to see. Always in some way or other, Dr. Shattuck cheered up the ward when he entered it. There was a greeting for every patient and a fund of stories that seemed to fit each case, some proper and some not quite so but always very much to the point. Many times I have watched him regarding with great amusement the mounting blush on the nurse's face as he told one of his stories which was a bit off color, and likewise the smile that lit up the patient's face.

House officers came and went but year after year these visits of Dr. Shattuck's continued and left an indelible impression on those of us who were lucky enough to be on his service. One of my cherished memories of this man happened late in his life shortly after I had moved into a little house in Brookline not far from Dr. Shattuck's own home where he lived after he

had retired from active practice. A wonderful basket of peaches from his own trees was left at my door with a note, "I don't believe that your own trees have had a chance to grow yet, so I am sending you some of my own." A little thing but characteristic of the man.

The science of medicine has made wonderful strides forward since the days of those men for whom I worked and knew so well, but the art of medicine which came from the heart as well as the brain as practiced by those men is rarely, if ever, equalled today.

COMPARISON OF DISEASE INCIDENCE IN CONNECTICUT WITH 1934 AND SEVEN YEAR AVERAGE

MONTH ENDING OCTOBER 12, 1935

	1935					1934			
Diseases	Week ending Sept. 21	Week ending Sept. 28	Week ending Oct. 5	Week ending Oct. 12	Average cases reported for week corresponding to Oct. 12 for past seven years	Week ending Sept. 22	Week ending Sept. 29	Week ending Oct. 6	Week ending Oct. 13
Amebiasis	—	—	—	—	—	1	—	1	—
Chicken Pox	9	15	16	16	23	14	30	43	22
Conjunctivitis Infectious	1	—	1	—	—	1	—	—	—
Diphtheria	2	3	5	7	8	3	—	—	2
Dysentery Bacillary	18	20	3	2	—	2	5	2	1
Encephalitis Epidemic	—	—	—	—	—	—	—	—	1
German Measles	—	2	5	7	2	—	5	3	1
Influenza	—	3	1	2	6	3	1	3	1
Malaria	1	—	1	—	—	—	—	—	—
Measles	9	9	31	37	12	9	17	17	18
Meningococcus Meningitis	—	—	—	1	—	—	1	—	1
Mumps	2	7	16	11	12	4	9	15	9
Paratyphoid Fever	1	2	—	—	—	—	1	—	—
Pneumonia (Broncho)	7	11	3	8	12	8	14	12	9
Pneumonia (Lobar)	8	9	11	8	17	10	7	10	8
Poliomyelitis	32	33	22	18	9	—	1	—	—
Scarlet Fever	37	13	27	24	22	8	9	11	16
Streptococcus Scre Throat	1	1	—	1	1	—	1	1	—
Tetanus	—	—	1	—	—	—	1	1	—
Trichinosis	—	—	—	—	—	—	—	—	1
Tuberculosis (Pul.)	26	21	40	19	21	27	25	34	14
Tuberculosis (O. F.)	2	2	1	—	1	—	2	1	2
Typhoid Fever	6	5	2	1	4	3	1	—	—
Undulant Fever	1	1	1	1	—	—	3	3	—
Whooping Cough	36	33	26	16	31	34	32	53	34
Gonorrhea	35	28	59	24	30	63	43	30	68
Syphilis	55	39	70	39	38	36	48	45	44

Remarks: No cases of Asiatic cholera, glanders, plague or yellow fever during the past seven years.

CASE RECORDS
of the
MASSACHUSETTS GENERAL HOSPITAL

ANTE MORTEM AND POST MORTEM RECORDS AS USED IN WEEKLY CLINICAL-PATHOLOGIC EXERCISES

FOUNDED BY RICHARD C. CABOT, M.D.

TRACY B. MALLORY, M.D., *Editor*

CASE 21441

PRESENTATION OF CASE

A ten months old baby girl was admitted because she had passed a bloody stool two weeks previously.

The patient was born at full term. There were no convulsions or any other postnatal disturbances. The child was breast fed for two months and since then had been on an adequate diet. She had received cod liver oil and orange juice every day since the second month, and cereals and puréed vegetables since the sixth month.

Approximately two weeks before entry the patient passed a large dark red stool. Tarry stools were passed that day and the following day, but the feces were not increased in amount. Following this incident she became irritable, weak and appeared to lose her appetite. She became quite pale. There was no history of vomiting, diarrhea, foreign body ingestion or jaundice.

Physical examination showed a well-developed and nourished, extremely pale infant who was quiet and observant. The skin had a slight icteric tint. There were no purpuric spots seen on the skin or mucous membranes. There were no hemangiomata apparent. The mucous membranes were very pale. The lungs were negative. The heart was not enlarged; the sounds were strong and there was a loud blowing systolic murmur over the entire precordium. The abdomen was soft. The liver edge could be felt just below the costal margin. The spleen was palpable but not very large.

The temperature was 100.8°, the pulse 160. The respirations were 34.

Examination of the urine was negative. The blood showed a red cell count of 1,760,000, with a hemoglobin of 30 per cent. The white cell count was 14,950. A smear showed achromia and variation in the size and shape of the red blood cells. The bleeding time was normal. The clotting time by capillary tube method was normal. Six stool examinations showed strongly positive guaiac tests. The stools were fairly well formed and brownish in color. There was little or no mucus present. No amebae could be found.

On the evening of admission she was given a transfusion of 150 cubic centimeters of citrated blood following which she showed marked improvement in spite of a mild transfusion reaction. Iron ammonium citrate 25 per cent, two drams twice a day, was given. On the third day she had a large formed stool; the center was gray and was surrounded by reddish black material. For the next two days the stools were tarry. Iron by mouth had been discontinued. Proctoscopic examination was negative. She was given another blood transfusion on the fourth day after her second hemorrhage. Her red blood cell count on the sixth day was 4,100,000, with a hemoglobin of 52 per cent. The white cell count was 29,500. The child at no time seemed to have acute pain. At times she seemed to be suffering from mild abdominal discomfort, to judge from her irritability and crying. She did not vomit while in the hospital. Her temperature was normal throughout the preoperative course in the hospital, except on admission and for a few hours following each of the two transfusions.

X-ray examination of the gastro-intestinal tract after a barium feeding was negative. A flat abdominal film was negative.

Approximately two weeks after entry, operation was performed.

DIFFERENTIAL DIAGNOSIS

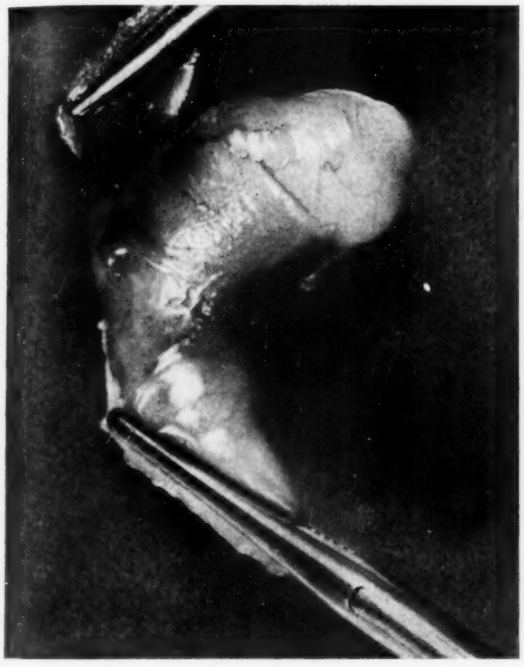
DR. THOMAS H. LANMAN*: A discussion of bleeding by rectum in this age group must include a number of conditions. From the history and physical examination we can rule out anything of an inflammatory nature such as ulcerative colitis which is rare, although we have seen cases under a year in our hospital. I think that tuberculosis can also be ruled out. There is, also, the question of some blood disease—one of the purpuras. That again I think can be ruled out by the normal clotting and bleeding time, the absence of purpuric spots and also to some degree by the suddenness of the onset. Another cause would be polyps in the intestinal tract. These are commonly found in the region of the rectum and it is unusual for them to give evidence of such massive hemorrhage as this child has had, or to produce a red count as low as that, although we have seen cases of long duration in which the red count dropped pretty low. Intussusception high in the intestinal tract does occur, but is quite rare and also is quite unlikely to give signs of sudden and massive hemorrhage. A large amount of blood loss such as she had on the first occasion is against intussusception. She is in the age group when the so-called idiopathic type of intussusception occurs. In our cases of this type of intussusception about 90

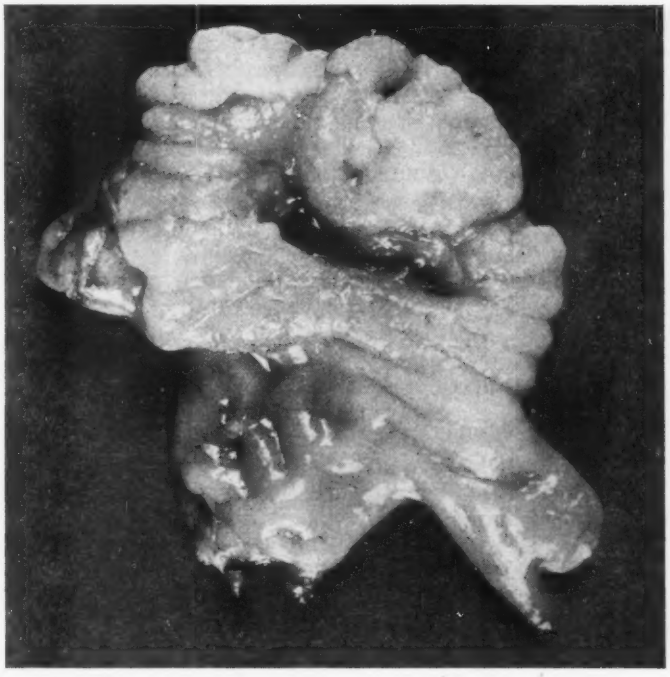
*Associate Visiting Surgeon, Children's Hospital, Boston.

per cent occur under a year. A point against its being a polyp is the fact that the rectal type of bleeding is much more likely to result in blood on the outside of the stool rather than mixed with it. In intussusception it would be very unusual for her to have as massive hemorrhage as this without the clinical symptoms characteristic of acute intestinal obstruction. We do get subacute or chronic forms of intussusception in this age group, but here, while bleeding does occur it is usually small in amount. Bleeding in the acute type is a result of marked interference with return of circulation. I do not believe you can get as much bleeding as this without pretty obvious signs of obstruction. It would be extremely unusual for intussusception to go on for the two weeks with that amount of hemorrhage and not have pretty obvious signs. The x-ray, moreover, is against it.

I think my guess would be that she probably bled from a Meckel's diverticulum. This occurs much more frequently than we formerly thought. We have seen a large number of cases in the past two years, the youngest two months of age, where bleeding by rectum was a presenting sign. In our series of forty-five Meckel's diverticula we have seen bleeding in seventy per cent of the cases, and in a great many of these the hemorrhage has been quite massive in amount. Recently we have found the red count below two million in a case where there was a perforation of an ulcer in a Meckel's diverticulum. I doubt if this case was perforated. The white cell count was 30,000, which might suggest diverticulitis associated with it. I doubt if there was perforation because I do not find any sign of peritonitis or of any obstruction. The discomfort that she complains of may be due to blood in the bowel itself. We have had cases where the blood was known to give a palpable mass. It is not uncommon for a Meckel's diverticulum to be the advancing point of an intussusception, but if intussusception were present in this case I should expect it to be incidental. I should expect to find a bleeding Meckel's diverticulum.

DR. HAROLD L. HIGGINS: We felt quite definitely that the blood was not coming from the large bowel because of the absence of mucus. We also were inclined to feel that the child did not have a duodenal ulcer. Babies do get duodenal ulcers and I have seen several such cases. They usually are in undernourished children, and the bleeding is scant rather than profuse.





PREOPERATIVE DIAGNOSIS

Ulcer of Meckel's diverticulum.

DR. THOMAS H. LANMAN'S DIAGNOSIS

Bleeding from Meckel's diverticulum.

PATHOLOGIC DIAGNOSIS

Ulcer of Meckel's diverticulum.

PATHOLOGIC DISCUSSION

DR. TRACY B. MALLORY: At operation a typical Meckel's diverticulum was found. The two clamps in the photograph are placed upon the ends of the resected segment of ileum and the short and rather fat diverticulum projects between them. The second picture shows the inside

of the diverticulum after it had been opened. The deeply punched-out linear ulcer is very evident and also the difference between the thick velvety mucosa lining the diverticulum and the thinner, glossier mucosa of the ileum can be distinguished. The reason for this difference in gross appearance became at once evident when the microscopic sections were examined. The mucosa in the diverticulum was very complicated in structure showing an irregular interadmixture of gastric and upper intestinal types of epithelium, the latter, however, predominating. The ulcer could be easily shown to have formed in the areas of intestinal mucosa rather than of gastric.

This case is very typical of many that have been reported. When ulcers are present in Meckel's diverticulum gastric mucosa of the fundic type can always be found, but the ulcers occur in the neighboring islands of intestinal epithelium. The interpretation of the pathogenesis as true peptic ulcer formation from the erosive action of gastric secretion unneutralized by bile, pancreatic juice, the secretions of Brunner's or pyloric glands can hardly be questioned and the evidence presented by these cases has, of course, been used by Lindau* and others as a strong inferential argument for the theory of hypersecretion as the basis of ulcer formation in the stomach and especially the duodenum.

The ulcer in this case was histologically typical of the active stage of peptic ulcer as one sees it in either stomach or duodenum or in the jejunum following gastroenterostomy. The so-called "fibrinoid necrosis", a brilliantly eosinophilic hyaline layer at the base of the ulcer which Buchner has emphasized was particularly well marked. At its deepest point the ulcer had penetrated the muscularis and only a millimeter of slightly thickened serosa lay between the base of the ulcer and the peritoneal cavity.

DR. LANMAN: When Dr. Henry Hudson looked up our cases he found gastric mucosa in seventy per cent, which was quite striking to us. I suppose we must have missed a lot. They go into collapse with sometimes a very massive hemorrhage, like a gastric ulcer. I was interested in looking it up and found that frequently in the last two or three years the red cell count had been below three million.

DR. HIGGINS: I referred to Abt's System of Pediatrics, published about 1927 and found there no mention of bleeding from a Meckel's diverticulum. The recognition of this condition is recent, certainly within the last fifteen years.

A PHYSICIAN: Is this condition ever seen in adults?

DR. MALLORY: Yes, several have been reported by Lindau.

*Lindau, A., and Wulff, H.: The peptic genesis of gastric and duodenal ulcer. Surg. Gyn. Obst. 53: 621 (Nov.) 1931.

CASE 21442

PRESENTATION OF CASE

A fifty year old white married American attorney entered complaining of pain in the left lower quadrant, and constipation.

Six years before entry he became ill with a vague aching pain in his left lower quadrant, localized for the most part midway between the left anterior superior iliac spine and the umbilicus. This was accompanied by loose watery stools. An x-ray at this time was reported as showing a large diverticulum which was causing obstruction. An operation was done following which there remained a fecal fistula. This healed spontaneously after about three months but one year later it reopened. Because of a suspected chest condition no surgical intervention was advocated. He was hospitalized, however, and with palliative care the fistula again healed. The suspicion of pulmonary disease was not confirmed. One and a half years later the patient went to his physician voluntarily for instruction in bowel management and a general check-up. Thereafter he remained quite well with only slight abdominal distress concomitant with upper respiratory infections. Four months before admission he again developed rather constant dull aching pain in the left lower quadrant, somewhat more severe than that of the initial episode. A protrusion of the operative scar appeared and gradually increased in size. Two months later he began to have pain in the region of the bladder and also in the right upper quadrant. This discomfort became progressively greater in intensity and particularly sharp just prior to bowel movement. One month previous to his entry he became nauseated and vomited everything ingested. This continued for two days and thereafter he remained well for three weeks, when he had another attack of vomiting which again persisted for two days. His bowel movements throughout his illness were irregular to the extent that he frequently went one or two days without going to stool. At times he had one or two scanty movements consisting of hard formed stools which were followed by a large soft formed stool. His appetite became quite poor during the last part of his illness. He had a great deal of flatus and complained of feeling "gurgling" in his abdomen.

At twenty years of age a septal operation was performed and five years later a double herniorrhaphy was done. He had "chronic bronchitis" for years which became much worse during the winter.

His father died at seventy-eight of prostate trouble. His mother died of liver disease at the age of fifty-five. One brother died of tuberculosis.

Physical examination showed a well-developed

and nourished middle-aged man who seemed to be quite comfortable. There was pyorrhea of the lower teeth and the upper teeth were absent. The heart and lungs were negative. The blood pressure was 130/80. In the left lower quadrant there was a small scar two centimeters in diameter which protruded so that a projecting mass about the size of a tennis ball was formed. This mass was very tender and loops of bowel were felt within it. The abdomen was held moderately rigid and tenderness was elicited generally with deep pressure. In the left lower quadrant only moderate pressure produced pain. The remainder of the examination was negative.

The temperature was 99.6°, the pulse 95. The respirations were 23.

Examination of the urine was negative. The specific gravity was 1.028. The blood showed a red cell count of 5,390,000, with a hemoglobin of 80 per cent. The white cell count was 18,900. The stools gave a positive reaction to the guaiac test. A Hinton test was negative. The nonprotein nitrogen of the blood was 45 milligrams per cent.

The patient remained quite comfortable for the first few days in the hospital. On the fourth day he began to vomit everything ingested. The tenderness in the lower abdomen increased and the temperature rose to 101.5°. He improved somewhat after this but since a mass was then palpated in the region of the old operative scar a surgical approach was deemed advisable. One week after admission under gas and ether anesthesia a partial repair of the incisional hernia and incision and drainage of a peritoneal abscess were done. On the following morning the patient went into profound shock with a thready pulse of 160 and a blood pressure of 88/70. Despite intravenous injections and a whole blood transfusion the patient did not rally and died on the ninth hospital day.

Differential Diagnosis

Dr. Horace K. Sowles: "An x-ray was reported as showing a large diverticulum which was causing obstruction." That is what we might expect from the clinical symptoms.

We presume that operation was a laparotomy with drainage of an abscess or drainage of the inflamed area due to perforation of a diverticulum.

We have as yet no indication whether the protrusion of the operative scar was a hernia, or possibly a recurrence of an abscess, except that he did not give any story of increased temperature at that time.

Pain in the bladder of course is consistent with an inflammatory process in the pelvis. The pain in the right upper quadrant is not so easily explained except that it is well known that obstruction of the left colon often causes extreme distention in the cecum and in the right colon.

"He had a great deal of flatus and complained of feeling 'gurgling' in his abdomen." I think that particular sort of complaint is pathognomonic of interference in the lumen of the intestinal tract. It is consistent with chronic obstruction.

The examination of the protruding mass in the left lower quadrant seems to rule out a superficial abscess since it contained loops of bowel.

Our findings still seem to point to an inflammatory process more in the pelvis and left lower quadrant but somewhat throughout the rest of the abdomen.

The temperature is not high, but the pulse is abnormally high and here we might bring out the point that the pulse in acute abdominal conditions or in intestinal obstruction is often a much better indicator of the patient's reaction or toxic condition than the temperature.

The white blood count is exceedingly high, which would seem to be evidence of inflammatory reaction.

Evidently it was felt now that it was necessary to drain what I assume to be an abscess. Of course we cannot rule out malignancy even though he had a diverticulitis six years ago. He may have continued to have an inflammatory process in his sigmoid since that time, but we cannot absolutely rule out a malignant disease. He has had no gross bleeding although the guaiac test is positive at the present time.

Twenty-four hours is a little late for what we consider acute surgical shock. Rapid pulse and low blood pressure might suggest hemorrhage but it is hard to conceive of a massive hemorrhage from this condition. I should think it would be more apt to indicate a fulminating and acute peritonitis secondary to the drainage of the pelvic abscess.

Although we cannot rule out malignant disease I think this patient had a primary diverticulitis with intermittent intestinal obstruction and an inflammatory process in his pelvis, or that secondary to malignant disease he developed a pelvic abscess which was drained; and I should assume that as a result of the operative procedure he developed acute fulminating peritonitis which was the cause of his pronounced shock.

Dr. Tracy B. Mallory: Dr. McKittrick, will you tell us what you found at operation?

Clinical Discussion

Dr. Leland S. McKittrick: We assumed that he had a recurrence of an old diverticulitis. He had a perfectly definite hernia in the old scar with the palpable loops of bowel as described here, moderate obstruction clinically, and he had obviously a fairly extensive inflammatory process. We were disturbed about

the recurrence of what we assumed to be his diverticulitis in relation to his old hernia because that made it pretty complicated to get at. He quieted down pretty well at first but when he flared up again it seemed imperative that we drain what we assumed to be an abscess. We went in medially to the old scar, not because we wanted to but because that was the point where he persistently had his maximum tenderness. Posteriorly there was very little tenderness that one could make out. While the desire was to go in lateral to the scar and posteriorly, we went in medially because we could not overlook his maximum tenderness. That took us directly into the abdominal cavity. Why he had the tenderness there, I do not know. We had gone over him a number of times and it had persisted. The abdomen was then carefully walled off and a large abscess lateral to the old scar and pointing at its posterior margin was drained from behind. It was simply necessary to break the adhesions posterior to the hernia, where we drained a large abscess. Nothing else was done but proximate the wound to a slight degree and he was given what was considered to be ample drainage.

DR. SOWLES: You did not do what was suggested here, a left hernia repair?

DR. MCKITTRICK: No, to close the wound it was necessary to free the edges up a little bit.

CLINICAL DIAGNOSES

Diverticulitis of the sigmoid with abscess.
Peritonitis?

DR. HORACE K. SOWLES' DIAGNOSES

Diverticulitis with abscess or malignant disease with abscess.
Acute peritonitis.

ANATOMIC DIAGNOSES

Multiple diverticula of the descending colon and sigmoid.
Acute diverticulitis.
Peritonitis, acute, generalized.
Pulmonary congestion and edema.
Atelectasis, partial, lower lobes.
Cyst of the right kidney, solitary, congenital.
Abscess, left flank.
Operative incisions: Exploratory laparotomy; incision and drainage of flank abscess.
Operative scar: Bilateral hernioplasty.
Arteriosclerosis, aortic, moderate.
Perisplenitis.

PATHOLOGIC DISCUSSION

DR. TRACY B. MALLORY: What was found at postmortem was a very extensive diverticulosis of the descending colon and sigmoid. The great majority of the diverticula were entirely free from any inflammatory change, but in a localized zone at just the upper end of the sigmoid there were numerous obviously inflamed diverticula on all sides of the gut. The omentum had become firmly adherent to the exposed peritoneal surfaces of some of these and this mass of thickened omentum about the large bowel formed a pretty considerable tumor mass in the left lower quadrant.

I presume the point of tenderness overlay one of these diverticula which had been successfully walled off by the omentum. Another diverticulum on the posterior surface had actually perforated and a frank abscess was found in the retroperitoneal tissues, into which led the drain which Dr. McKittrick had inserted. We found a very slight but diffuse general peritonitis. All of the serosal surfaces were reddened and covered with a very thin film of fibrin. There was nothing else that could account anatomically for death, so I am inclined to agree with Dr. Sowles' assumption that a very acute peritonitis was probably the most significant immediate factor at death. It would not be possible anatomically, of course, to rule out an element of shock.

A PHYSICIAN: When the first exploratory incision was made was the possibility considered of closing that and making a second incision laterally?

DR. MCKITTRICK: Because of the hernia the wound could not be closed without freeing the bowel and omentum laterally and this brought us into the abscess.

DR. SOWLES: I should like to ask if he had anything to account for the right-sided pain?

DR. MCKITTRICK: No; I was not over on the right side at all.

A PHYSICIAN: Is it possible to drain them by a retroperitoneal operation rather than opening the peritoneal cavity?

DR. MCKITTRICK: Yes. I think frequently you can get in from behind but you have to have some lead. You cannot go in unless there is tenderness or something there that would lead you to make that incision.

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STERILIZATION AND THE PSYCHOSES

THE rapid increase of mental disease in this country has been the subject of many recent articles. It has been stated that the birth rate of people with subnormal intellects is so much greater than that of normal people that racial degeneration is inevitable. One way of solving this problem is to sterilize the unfit. Is such a procedure to be advised?

This question has been studied by a committee appointed in May, 1934, by the American Neurological Association, assisted by a grant from the Carnegie Foundation. The committee, consisting of Doctor Abraham Myerson, Chairman, Doctors J. B. Ayer, Tracy J. Putnam, Clyde E. Keeler and Leo Alexander, has recently published a preliminary report. The conclusions are at such variance with the popular ideas on this subject that they deserve careful consideration.

The committee states, to begin with, that there is no valid evidence that mental disease and mental defects are on the increase. Commitment to hospitals and asylums is increasing, but this, as the committee points out, is a factor which operates against propagation. The mentally sick, diseased and defective show less tendency to marry and among those who do marry the birth rate is low.

The contributions bearing on heredity made by the study of genetics do not support the contentions of the eugenists, for research has shown, according to Riddle, that "the specific conditions under which a gene or factor operates and develops have an equal value with the germinal factors in the appearance of anything that can be called heredity. . . . Heredity is controlled whenever and wherever an aspect of development is controlled." The committee goes on to state: "His whole thesis, which seems to us to be extremely pertinent to the problems of psychiatry, is that by manipulating both the external environment and the internal environment of the individual, first, the characters of the individual may be altered from their otherwise normal hereditary pattern; more importantly, that there is work to show that the germplasm itself may be modified."

Only two psychoses have a hereditary significance—manic-depressive and dementia praecox. Of the two, the former is more definitely hereditary. Factors of heredity exist in feeblemindedness, but it is not a simple recessive Mendelian character.

Interesting experiments have been carried out as to the relative values of heredity and environment in relation to intelligence. Some of these experiments, summarized in the committee's report, appear to prove quite conclusively that environment, especially in the early years of life, plays an important part in the development of the child's intelligence.

As regards the inheritability of epilepsy, the committee accepts the statement that epilepsy occurs more frequently in the descendants of epileptics than in the descendants of non-epileptics, but does not believe that there is any single recessive Mendelian character which can explain its incidence.

The committee comments favorably on Lange-Eichbaum's study of genius and heredity. "Enough evidence is adduced, . . . to indicate that sterilization might readily cut off from the race some of its most valued and valuable members. . . ." Concerning the inheritance of criminal tendencies, most writers agree that while there may be a constitution, the effort to breed it out by any eugenical measures is in the present state of our knowledge not to be recommended.

In concluding its study of sterilization, the committee expresses its views as follows:

"*First:* Our knowledge of human genetics

has not the precision nor amplitude which would warrant the sterilization of people *who themselves are normal* in order to prevent the appearance, in their descendants, of manic-depressive psychosis, dementia praecox, feeblemindedness, epilepsy, criminal conduct or any of the conditions which we have had under consideration. An exception may exist in the case of normal parents of one or more children suffering from certain familial diseases, such as Tay-Sachs amaurotic idiocy.

Secondly: Particularly do we wish to emphasize that there is at present no sound scientific basis for sterilization on account of immorality or character defect.

Thirdly: Nothing in the acceptance of heredity as a factor in the genesis of any condition considered by this report excludes the environmental agencies of life as equally potent and, in many instances, as even more effective."

As a result of these conclusions, the committee recommends that any law concerning sterilization passed in the United States under the present state of knowledge should be voluntary and regulatory rather than compulsory, and should apply to patients in private institutions and at large as well as to those in State institutions. Other recommendations are made concerning the machinery for administering sterilization laws, after which the committee states that it can recommend sterilization only in selected cases of certain diseases and with the consent of the patient or of those responsible for him. These diseases, arranged in the order in which sterilization would appear to be indicated, are as follows:

"(1) Huntington's chorea, hereditary optic atrophy, familial cases of Friedreich's ataxia, and certain other disabling degenerative diseases recognized to be hereditary.
(2) Feeblemindedness of familial type.
(3) Dementia Praecox (schizophrenia).
(4) Manic-depressive psychosis.
(5) Epilepsy."

The report emphasizes the need for further research along this line, and recommends the appointment of a permanent committee for this purpose. The concluding paragraph summarizes the views of the committee on the present status of sterilization.

"Furthermore, it is to be emphasized that no great or radical change in the complexion of society can be expected from any such sterilization program as we recommend, nor from any justifiable legislation. We do not believe that society needs to hurry into a program based on fear and propaganda. Although the problem of mental disease and defectiveness is enormous, there is no new social or biological emergency."

NUTRITION AND DEPRESSION

A STUDY of 5,000 city children between the ages of six and nine years, made during five years of depression by the United States Public Health Service, shows that the weight of those from depression-poor families has inclined definitely downward.

These children were divided into three groups: those whose families had remained in comfortable economic circumstances; those whose families remained poor, and those whose families, originally comfortable in 1929, had become poor during the depression. The relative weight line for the first group remained approximately level, the line for the second group inclined definitely downward, and the line for the third group inclined somewhat upward—which reminds one of the old saying that God takes care of the poor and the rich take care of themselves.

THIS WEEK'S ISSUE

CONTAINS articles by the following named authors:

DAYTON, NEIL A. M.D. Ohio State University College of Homeopathic Medicine 1915. Director, Division of Mental Deficiency; Division of Statistics; Rockefeller Research Project in Mental Diseases and Defect, Massachusetts Department of Mental Diseases. Instructor in Psychiatry, Tufts College Medical School. His subject is "A New Method of Calculating Discharge Rates in Mental Diseases with Special Consideration of the Age Factor." Page 841. Address: Room 167, State House, Boston, Mass.

GOLDWATER, LEONARD J. A.B., M.D. New York University, University and Bellevue Hospital Medical College 1928. Instructor in Medicine, New York University College of Medicine. Clinical Assistant Visiting Physician, Third (N. Y. U.) Medical Division, Bellevue Hospital. Director, Student Health Service, New York University College of Medicine. Address: 53 East 82nd Street, New York City. Associated with him is

STEINBERG, ISRAEL. A.B., M.D. Harvard University Medical School 1928. Assistant Visiting Physician, Third (N. Y. U.) Medical Division, Bellevue Hospital. Instructor in Medicine, New York University College of Medicine. Address: 103 East 84th Street, New York City. And

MOST, HARRY. B.S., M.D. New York University, University and Bellevue Hospital Medical College 1931. Clinical Assistant Visiting Physician, Third (N. Y. U.) Medical Division, Bellevue Hospital. Assistant in Medicine, New York University College of Medicine. Address: London School of Tropical Medicine, Keppel Street, London, W.C.1, England. And

CONNERY, JOSEPH E. M.D. New York University, University and Bellevue Hospital Medical College 1914. Associate Professor of Clinical Pathology, New York University College of Medicine. Visiting Physician, Third (N. Y. U.) Medical Division, Bellevue Hospital. Address: 75 East 55th Street, New York City. Their subject is "Hemoptysis in Trichiniasis." Page 849.

KING, M. K. M.D. Medical College of Virginia 1930. Passed Assistant Surgeon, United States Public Health Service. In charge of Surgical Service, United States Marine Hospital, Savannah, Georgia. His subject is "Uncinariasis and Appendicitis." Page 851. Address: United States Marine Hospital, Savannah, Ga.

COGAN, DAVID G. A.B., M.D. Harvard University Medical School 1932. Clinical Assistant in Ophthalmology, Massachusetts Eye and Ear Infirmary. Assistant in Ophthalmology, Harvard University Medical School. Address: 81 Phillips Street, Boston, Mass. Associated with him is

COGAN, FRANCES C. A.B., M.D. Johns Hopkins University School of Medicine, 1934. Address: 81 Phillips Street, Boston, Mass. Their subject is "Cataracts and Dinitrophenol." Page 854.

WHITAKER, LESTER R. M.D. Harvard University Medical School 1923. F.A.C.S. First Assistant Visiting Surgeon, Massachusetts Memorial Hospitals, Boston. Associate Member, Evans Memorial for Clinical Research and Preventive Medicine. Instructor in Surgery, Boston University School of Medicine. His subject is "Electrosurgical Appendectomy." Page 856. Address: 41 Bay State Road, Boston, Mass.

FRENCH, RALPH W. A.B., M.D. Harvard University Medical School 1910. F.A.C.S. Surgeon, Truesdale Hospital, Fall River, Mass. His subject is "Primary Abscess of the Omentum." Page 857. Address: 151 Rock Street, Fall River, Mass.

ATCHLEY, DANA W. S.B., M.D. Johns Hopkins University School of Medicine 1915. Associate Professor of Medicine, Columbia University College of Physicians and Surgeons. Associate Visiting Physician, Presbyterian Hospital, New York City. His subject is "The Rôle of Peripheral Circulatory Failure in Clinical Medicine." Page 861. Address: Presbyterian Hospital, 620 West 168th Street, New York City.

GALDSTON, IAGO. B.S., M.D. Fordham University School of Medicine 1921. Secretary, Medical Information Bureau, New York Academy of Medicine. Consultant in Health Education, National Tuberculosis Association. His subject is "The Economic and Social Aspects of Socialized Medicine." Page 868. Address: 2 East 103rd Street, New York City.

HAWES, JOHN B., 2ND. A.B., M.D. Harvard University Medical School 1903. Formerly, Assistant Visiting Physician, Massachusetts General Hospital; Director, Pulmonary Clinic and Non-Pulmonary Clinic, Massachusetts General Hospital; Consultant, Diseases of the Lungs, New England District, United States Veterans Bureau; and Secretary, Massachusetts Tuberculosis Commission. Now, President, Boston Tuberculosis Association; Director, Massachusetts Tuberculosis League; Rutland Cottage Sanatoria and National Tuberculosis Association. Consultant, Beth Israel Hospital; Jordan Hospital, Plymouth; and Henry Heywood Memorial Hospital. His subject is "Memories of a Great Physician, Dr. Frederick C. Shattuck, of Boston." Page 873. Address: 330 Dartmouth Street, Boston, Mass.

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C. J. KICKHAM, M.D., *Chairman*, 524 Commonwealth Ave., Boston, Mass.
R. S. TITUS, M.D., *Secretary*, 472 Commonwealth Ave., Boston, Mass.

THE TREATMENT OF VOMITING OF PREGNANCY

There is probably an underlying physiologic disturbance as a basis for all vomiting of pregnancy, although the importance of superimposed psychic disturbances in many cases is not to be denied. So-called "toxic" vomiting probably does not differ from "non-toxic" vomiting except as regards the severity and persistence of emesis. The pathology of the disease may be adequately accounted for on the basis of starvation alone, provided one includes deficiency of vitamines with the water, mineral, and other food deficiencies of starvation.

For the purpose of outlining treatment, patients who vomit may be arbitrarily divided into three groups as follows:

I. *"Physiologic"* vomiting

The nausea and morning vomiting which frequently occur for a few weeks after the first missed period require no especial treatment other than reassurance, mild doses of sedatives in apprehensive patients, and the suggestion that the patient take a glass of milk or cup of coffee some twenty minutes before arising in the morning.

*A series of short selected articles by members of the Section will be published weekly.
Comments and questions by subscribers are solicited and will be discussed by members of the Section.

II. *Mild Hyperemesis* includes patients who persistently vomit a significant portion of the normal food and fluid intake. However, they retain enough food so that clinical dehydration, marked weight loss, and acidosis (i.e., persistent acetone and diacetic acid in the urine) are absent.

In the treatment of these patients, intelligent management of the frequent causes of psychic disturbances in the home is often an important factor. Disturbing elements in the environment should be removed when possible. It is advantageous temporarily to dispose of "helpful" friends and relatives. Excitement should be avoided, and the mind occupied with matters other than the vomiting. Moderate doses of barbiturates (i.e., phenobarbital gr. ¾ to 1½ B.I.D.) aid in the control of nervousness, and seem to allay nausea. The bowels should be kept open with mild catharsis or enemata. The nausea often seems to be brought on or aggravated by hunger, hence six small meals daily instead of the three usual ones are helpful. The elimination of fats from the diet temporarily is also helpful. The diet should be easily digestible, rich in vitamines and minerals, as well as adequate in carbohydrate and protein. Empirically, many patients seem to improve after liberal doses of vitamine B complex. Coated tablets of Brewer's yeast concentrate are made by several pharmaceutical houses.

Most patients will respond to these measures. Those who do not improve promptly should be watched daily for signs of dehydration and acidosis.

III. *Severe Hyperemesis*

This group includes patients who retain less than half of the normal diet and show marked weight loss, dehydration and acidosis. Mild cases of patients who vomit but who fail to respond to a few days' treatment as outlined above, and those whose vomiting is severe when first seen should be removed to a hospital.

On admission the patient should be isolated, and no visitors allowed until vomiting has been controlled. She should be left alone except for strictly necessary visits of physician and nurse. No emesis basin or equipment reminiscent of vomiting should be visible. The T.P.R. should be recorded every four hours, and the twenty-four hour urine volume measured. Admission and daily urine specimens should be examined routinely, particular attention being given to testing for acetone, diacetic acid and bile.

The initial physical examination should include careful inspection of the mouth for sore tongue and search for evidence of peripheral neuritis; findings which suggest vitamine B complex deficiency.

On admission the patient should have an enema following which a liberal dose of a barbiturate may be given by rectum (i.e., pentobarbital gr. iv ss). The deficient water, salt, and carbohydrate should be corrected immediately by hypodermoclysis of normal saline (2000-3000 cc.) and 10 per cent glucose solution, 500 cc. given slowly intravenously. Rectal taps sometimes aggravate nausea and vomiting. Saline clyses and intravenous glucose are repeated at frequent intervals in sufficient quantities to maintain a daily urine volume of over 1200 cc. free of acetone and diacetic acid. The pentobarbital is repeated in 3 grain doses at intervals of six to twelve hours. Nothing is offered the patient by mouth until vomiting has ceased for eighteen to twenty-four hours. She is then given fluids hourly, such as milk, ounces VI, orange juice ounces VI, and water ounces VI alternating for fourteen hours of the day. When the hourly fluids have been taken for two days without significant vomiting, the patient is offered frequent small meals of attractive easily digestible solid food. A daily bowel movement is important throughout treatment, and should be secured if necessary by enema.

An occasional patient will continue to vomit in spite of this régime. The responsibility for such patients, as well as for those whose condition is poor when first seen, should be shared with a competent obstetrician before the condition becomes desperate. Of particular significance in evaluating the state of affairs are the following signs: persistent pulse of 110 or over, persistence of acidosis, the appearance of bile in the urine, early signs of peripheral neuritis, mental confusion, and other peculiarities of behavior.

If by any means, a well-balanced nutritious diet with large doses of vitamine B complex can be given to such patients, therapeutic abortion is seldom indicated. Feedings through the duodenal tube by constant drip of a liquid diet reinforced with large doses of vitamine B complex have been successful in a group of such patients in whom other methods had failed. A Levine tube is passed through the nose after liberal sedation with a barbiturate, and manipulated into the duodenum if possible. The stomach is washed with dilute bicarbonate solution and a liquid diet is fed at body temperature through the tube by drip. Feedings are given hourly for twelve hours daily and the rate of the drip is regulated so that each feeding runs in in thirty minutes. The diet consists of milk ounces IV with a heaping teaspoon of Brewers' yeast concentrate (Harris), orange juice ounces IV sweetened with corn syrup, and eggnog ounces IV, alternating for twelve hours daily. The tube is withdrawn an hour after the last feeding, to be replaced the following morning. A small amount of the feeding is sometimes regurgitated at first but all patients as yet so treated have retained the bulk of the food. Feedings are continued through

the tube from two to five days depending upon the severity of the condition.

However, if a patient is encountered who is refractory to all measures, and therapeutic abortion is considered, it is important to get obstetrical consultation before the condition is desperate. Of patients who have progressed to the point of persistent rapid pulse, mental aberration or signs of peripheral neuritis, the mortality is high regardless of whether therapeutic abortion is performed.

It is advisable in the after-care of severe vomiters to give a rich source of vitamine B complex in addition to a well-balanced high vitamine diet for some weeks after vomiting has ceased. Also, although hemoglobin and red blood cell estimations are high during the vomiting due to blood concentration, many of these patients on recovery have a relatively severe secondary anemia for which the exhibition of iron is beneficial.

THIRD ANNUAL POSTGRADUATE MEDICAL EXTENSION COURSE

The following sessions have been arranged by the Committee for the week beginning November 3:

Barnstable

Sunday, November 3, at 4:00 P.M., at the Cape Cod Hospital, Hyannis. Subject: Lung Diseases: (a) Differential Diagnosis and Treatment of Lobar Pneumonia. (b) The Surgical Problems of Empyema. Instructors: J. H. Pratt and J. W. Strieder. J. I. B. Vail, Chairman.

Bristol South (Fall River Section)

Monday, November 4, at 4:00 P.M., at the Stevens Clinic of the Union Hospital, Prospect Street, Fall River. Subject: Pediatrics: The Neonatal State and its Diseases; Medical and Surgical Aspects. Instructors: W. R. Sisson and P. J. Mahoney. Eugene A. McCarthy, Chairman.

Essex North

Friday, November 8, at 4:00 P.M., at the Hotel Bartlett, 95 Main Street, Haverhill. Subject: Neurological Aids in the Diagnosis and Treatment of Disease from the Medical Viewpoint. Problems of History and Examination with Special Reference to (a) Neurosyphilis, Multiple Sclerosis and Other Degenerative Conditions. (b) Diseases with Acute Onset, such as Meningitis and Cerebral Accidents. Instructor: J. B. Ayer. Francis W. Anthony, Chairman.

Essex South

Tuesday, November 5, at 4:00 P.M., in the Nurses' Home of the Salem Hospital, Salem. Subject: Immunology: Latest Developments in Immunization. Smallpox, Typhoid, Measles, Scarlet Fever, Diphtheria, Whooping Cough, and Infantile Paralysis. Instructor: R. F. Feemster. Walter G. Phippen, Chairman.

Hampden

Thursday, November 7, at 4:00 P.M., at the Academy of Medicine, Professional Building, 20 Maple Street, Springfield, and at 8:00 P.M., at the Holyoke City Hospital, Holyoke. Subject: Cancer of Breast and Uterus; Sarcomas of Bone; Lymphoma and Leukemia, Their Early Diagnosis. Discussion of Life History of Cancer and Grades of Malignancy. Instructors: E. M. Daland and G. G. Smith. George L. Schadt and George D. Henderson, Chairmen.

Hampshire

Wednesday, November 6, at 4:15 P.M., in the Nurses' Home of the Cooley Dickinson Hospital, Northampton. Subject: Immunology: Latest Developments in Immunization. Smallpox, Typhoid, Measles, Scarlet Fever, Diphtheria, Whooping Cough, and Infantile Paralysis. Instructor: W. W. Lee. Robert B. Brigham, Chairman.

Middlesex East

Wednesday, November 6, at 4:00 P.M., at the Melrose Hospital, Melrose. Subject: Psychiatry: Management of Psychic States in the Care of General Diseases, Especially Chronic Disorders, Subpsychotic States. Instructors: G. C. Caner and Maurice Fremont-Smith. Joseph H. Fay, Chairman.

Middlesex South

Tuesday, November 5, at 4:15 P.M., at the Cambridge Hospital, Cambridge. Subject: Kidney and Bladder Diseases, A (Surgical). Hematuria: Its Significance in Surgical Diseases of Kidney and Bladder. Instructor: F. H. Colby, Edmund H. Robbins, Chairman.

Norfolk South

Monday, November 4, at 8:30 P.M., at the Quincy City Hospital, Quincy. Subject: Pediatrics: Abdominal Disease in Childhood; Medical and Surgical Aspects. Instructors: P. H. Sylvester and H. W. Hudson, Jr. David L. Belding, Chairman.

Plymouth

Tuesday, November 5, at 4:00 P.M., at the Brockton Hospital, Brockton. Subject: Pediatrics: Abdominal Disease in Childhood; Medical and Surgical Aspects. Instructors: J. L. Morse and W. E. Ladd. W. H. Pulsifer, Chairman.

Worcester North

Friday, November 8, at 4:30 P.M., at the Burbank Hospital, Fitchburg. Subject: Kidney and Bladder Diseases. B (Surgical). Prostatism and Related Diseases. Cystitis and Pyelitis. Instructor: B. C. Wheeler. Edward A. Adams, Chairman.

MISCELLANY

ANTERIOR POLIOMYELITIS CASES FOR 1935

WEEKLY LIST, OCTOBER 21-26

City or Town	
Fall River	2
Plymouth	1
Brockton	2
Dedham	1
Framingham	1
Boston	4
Cambridge	1
Chelsea	3
Lexington	1
Malden	1
Medford	2
Needham	1
Newton	1
Revere	1
Watertown	1
Andover	3
Peabody	1
Salem	2
Fitchburg	1
Hudson	1
Worcester	2
Holyoke	1
Springfield	1
	35

PHYSICIANS REGISTERED BY EXAMINATIONS HELD JULY AND OCTOBER, 1935

THE COMMONWEALTH OF MASSACHUSETTS
BOARD OF REGISTRATION IN MEDICINE
JULY 11, 1935

Kenneth L. Stout, Massachusetts General Hospital, Boston, Mass.
Ruth Burr, 56 Commonwealth Avenue, Boston, Mass.
Kerwin Kinard, Wenham-Hamilton, Mass.
John B. Dineen, Monson State Hospital, Palmer, Mass.
Felix M. Brown, 163 Hillside Avenue, Arlington, Mass.
Siegfried J. Thannhauser, 1101 Beacon Street, Brookline, Mass.
Hollis E. Vernon, 49 Cherry Street, Spencer, Mass.
Arthur B. Serino, 462 Putnam Avenue, Cambridge, Mass.
Nathan Epstein, Boston Floating Hospital, Boston, Mass.
Louis S. Chase, 140 Harvard Street, Everett, Mass.
Alphonse F. Popoli, 6 Bellevue Road, Quincy, Mass.
Arie C. van Ravenswaay, 198 Commonwealth Avenue, Boston, Mass.
Joseph G. Cutler, 43 Bow Street, Beverly, Mass.
Herbert M. Williams, New York Hospital, New York City.
Charles Meinhardt, 340 West Kinney Street, Newark, N. J.
Warren D. Thomas, Mercy Hospital, Springfield, Mass.
John B. Zielinski, 473 Hillside Avenue, Holyoke, Mass.
Jacob J. Klar, St. Luke's Hospital, Newburgh, N. Y.
Maxon H. Eddy, Middlebury, Vt.
Edmund L. Carey, 59 South Street, Quincy, Mass.
Gordon A. Saunders, 47 Horne Road, Belmont, Mass.
Timothy H. McSweeney, North Creek, N. Y.
Hans Fulder, Boston City Hospital, Boston, Mass.
Dana L. Farnsworth, 228 Main Street, Williamstown, Mass.
Edward N. Anderson, 27 Egmont Street, Brookline, Mass.
John D. Maloney, 31 Wave Avenue, Wakefield, Mass.
Philip Faraci, 30 Pilling Street, Haverhill, Mass.
John J. Cincotti, 39 Cooper Street, Boston, Mass.
George H. Boynton, 20 Grove Street, West Medford, Mass.
Folke W. Wiklund, 15 Moultrie Street, Dorchester, Mass.
Charles L. Buono, New Casualty Hospital, Washington, D. C.
James S. Stillman, Turkey Shore Road, Ipswich, Mass.
Hirsh W. Sulkowitch, Massachusetts General Hospital, Boston, Mass.
Charles H. Kelley, Beverly Hospital, Beverly, Mass.
Charles R. Lord, Jr., 83 High Street, Ipswich, Mass.
Frank A. Mahoney, Jr., 42 Crescent Avenue, Chelsea, Mass.
James J. Bradley, 43 Oak Avenue, Belmont, Mass.
Albert M. Bond, New England Sanitarium and Hospital, Melrose, Mass.
Oscar Feinsilver, 48 Jones Road, Beachmont, Mass.
Bernard B. Brass, 62 Orange Street, Chelsea, Mass.
Robert H. Atkins, 612 Merrick Avenue, Detroit, Mich.
Joseph H. Nicholson, 497½ Hampshire Street, Lawrence, Mass.
Sydney S. Deutch, 93 Watertown Street, Watertown, Mass.
Samuel B. Biller, 184 Orchard Street, Watertown, Mass.
Henry M. Bernhardt, 100 Kilsyth Road, Brighton, Mass.
Samuel M. Tarnower, 1573 East 10th Street, Brooklyn, N. Y.
Daniel J. Mullane, 71 Spring Park Avenue, Boston, Mass.
Edward A. Abbot, Mercy Hospital, Springfield, Mass.
Edward F. Lawlor, Jr., 33 Tremont Street, Lawrence, Mass.
William C. Gould, Jr., 63 Russell Street, Worcester, Mass.
John F. Healy, St. John's Hospital, Lowell, Mass.
Reuben H. Frogel, 187 Callendar Street, Dorchester, Mass.

William C. Carey, 178 Newton Street, Meriden, Conn.
Vlado A. Getting, 25 Charles Street, Dorchester, Mass.
Emerson A. Read, 55 Dixwell Avenue, Quincy, Mass.
Eugene F. Smith, St. Luke's Hospital, New Bedford, Mass.
Alice Ettinger, 25 Bennet Street, Boston, Mass.
Matthew J. Bachulus, 973 Main Street, Walpole, Mass.
William R. Hill, 108 Butler Avenue, Providence, R. I.
Robert Dutton, 33 Avon Street, Wakefield, Mass.
Ralph A. Ross, 1769 Commonwealth Avenue, Brighton, Mass.
Hyman Heller, 252 Main Street, Webster, Mass.
Francis H. Higgins, 9 Lord Street, Waltham, Mass.
Reginald S. Hunt, 80 East Concord Street, Boston, Mass.
Leo Koretsky, Springfield Hospital, Springfield, Mass.
Peter B. Hagopian, 50 Oak Street, Lawrence, Mass.
Jeremiah E. Greene, 85 Dudley Road, Newton, Mass.
William J. Devlin, 42 Arborway, Jamaica Plain, Mass.
Blanche C. Allen, 1401 State Street, Springfield, Mass.
Joseph J. Michaels, Boston Psychopathic Hospital, Boston, Mass.
Theophane M. Poirier, 15 Royal Street, Plymouth, Mass.
Robert McCastline, Northfield, Mass.
Michael Peters, Hahnemann Hospital, Philadelphia, Penn.
Arthur L. Wallace, 196 Main Street, Nashua, N. H.
Harry A. Warren, Peter Bent Brigham Hospital, Boston, Mass.
Harry Levine, 12 Grove Street, New York City.
Samuel F. Marshall, Henry Ford Hospital, Detroit, Mich.
Willard W. Wall, Norfolk County Hospital, Braintree, Mass.
Hugo V. Ascolillo, 19 Garden Street, Somerville, Mass.
Lawrence P. Bowser, 33 Cedar Street, Stoneham, Mass.
Raymond F. Haling, 146 East Main Street, Chicopee Falls, Mass.
Julius Rubin, 4 McLean Street, Boston, Mass.

OCTOBER 1, 1935

John W. Zeller, Massachusetts General Hospital, Boston, Mass.
Benjamin Spritz, 127 Shurtleff Street, Chelsea, Mass.
Wilmer C. Smith, c/o The Two Companies, Enfield, Mass.
Louis H. Cohen, Worcester State Hospital, Worcester, Mass.
Miriam J. Hosmer, 280 Washington Street, Woburn, Mass.
Jacob Brem, 16 Hansborough Street, Dorchester, Mass.
Samuel B. Kirkwood, 52 Garden Street, Cambridge, Mass.
Joseph L. Campbell, Cotuit, Mass.
Karl J. Thomson, 55 Lanack Road, Brighton, Mass.
Mandel E. Cohen, Massachusetts General Hospital, Boston, Mass.
John D. Stewart, Massachusetts General Hospital, Boston, Mass.
Joshua C. Drooker, Boston City Hospital, Boston, Mass.
Edwin J. Gillis, 16 Holland Avenue, Pittsfield, Mass.
Glin C. Hendrix, 249 River Street, Mattapan, Mass.
Medora V. Eastwood, Box 57, Plymouth, Mass.
Arthur J. Gavigan, Worcester State Hospital, Worcester, Mass.
Richard J. Alt, Beverly Hospital, Beverly, Mass.
Robert F. Cahill, 25 Division Street, Brockton, Mass.
John E. Cahill, 25 Division Street, Brockton, Mass.
Lewis C. Donahue, 3 Marshall Street, Needham, Mass.

NOTICES

LECTURES BY DR. S. J. THANNHAUSER

Dr. S. J. Thannhauser, formerly Professor of Medicine and Director of the Clinic of the University of Freiburg, Germany, is giving a course of clinical lectures on Saturday mornings at 9 A.M. at the Boston Dispensary. The subjects for November are as follows:

November 2—Diabetes
November 9—Diabetes.
November 16—Liver Disease.
November 23—Peptic Ulcer.

Practitioners in any part of New England are cordially invited. This course is made possible by a grant from the Bingham Associates Fund for the Advancement of Rural Medicine.

MEDICAL CLINIC AND STAFF ROUNDS AT THE PETER BENT BRIGHAM HOSPITAL

At 3:30 P.M. on Thursday, November 7, in the amphitheater of the Peter Bent Brigham Hospital, Dr. Henry A. Christian, Physician-in-Chief, Hersey Professor of the Theory and Practice of Physic in the Harvard Medical School, will give a medical clinic. To it are cordially invited practitioners and medical students.

On Saturdays in the wards of the Peter Bent Brigham Hospital, from 10 to 12, staff rounds will be conducted by Dr. Christian.

HARVARD MEDICAL SCHOOL COLLOQUIUM

Luncheon meetings of the Harvard Medical School Colloquium will be held again this year in the Bowditch Room of Vanderbilt Hall on Thursdays at 1 P.M. These meetings are for the purpose of bringing medical students and teachers into closer con-

tact with their own several departments and also with the University as a whole. The meetings in November will be as follows:

November 7—Studies of the Circulation of Pregnant Women. Dean C. S. Burwell.

November 14—The Ethiopian Situation. Dr. C. S. Coon.

November 21—Where Medicine is Going. Assistant Professor D. V. Brown.

MASSACHUSETTS DEPARTMENT OF EDUCATION

UNIVERSITY EXTENSION COURSE

Mental Hygiene for Adults
Gardner Auditorium, State House, Boston

This course offers an unusual opportunity to hear outstanding leaders in the mental hygiene movement discuss in simple non-technical language principles and mechanisms of mind that control our everyday lives. Rich in stimulating suggestions and valuable advice for better and wiser living, these lectures appeal universally. All, regardless of age, position, financial standing, or education, will be interested in the personal applications of these lectures. The course will be particularly suggestive for nurses, teachers, social workers, doctors and parents,—those directly responsible for the welfare of others.

The course is offered in coöperation with the Massachusetts Society for Mental Hygiene. It may be taken either (1) as a series of lectures which entails no class work but simply attendance at meetings, or (2) as a credit course which requires class work, collateral reading, and a final examination. Certificates will be awarded by the Massachusetts Department of Education to those who complete the requirements for the credit course.

PROGRAM

Course Leader:
Henry B. Elkind, M.D., Medical Director, Massachusetts Society for Mental Hygiene

November 5. Keeping Mentally Fit—Joseph Jastrow, Ph.D., formerly Professor of Psychology, University of Wisconsin.

November 12. The Management of Disease in Childhood in Relation to Mental Health — Bronson Crothers, M.D., Assistant Professor of Pediatrics, Harvard Medical School.

November 19. The Child and the Modern World—Lawson G. Lowrey, M.D., Director, Institute for Child Guidance, New York City.

December 3. How Psychiatry Can Aid in Meeting Problems of Modern Life—Donald Gregg, M.D., President, Massachusetts Society for Mental Hygiene.

December 10. Current Ethical Trends — James J. Walsh, M.D., Professor of Physiological Psychology, Cathedral College, and Extension Lecturer, Fordham University, New York.

December 17. Adult Problems: "The Blues" and Fatigue States—Karl M. Bowman, M.D., Associate Professor of Psychiatry, Harvard Medical School.

January 7. Psychoanalysis and Mental Health—Jacob Kasanin, M.D., Clinical Director, State Hospital for Mental Diseases, Howard, R. I.

January 14. Adults in Difficulty — A. Warren Stearns, M.D., Dean, Tufts College Medical School.

Register now.

This class will meet weekly on Tuesday evenings at 7:45 P.M., beginning November 5, 1935. Enrollment may be made at the University Extension Office, Room 217, State House, or at the first meeting of the class. Charge: $4 for auditors; $6 for credit students; text material extra.

NOTICES OF MEETINGS

BOSTON UNIVERSITY SCHOOL OF MEDICINE SURGICAL CLINIC AT THE BOSTON CITY HOSPITAL

Friday, November 1, 12-1, Cheever Amphitheatre. Dr. Irving J. Walker, Clinical Professor of Surgery, Harvard Medical School, will talk on "Jaundice." Physicians and medical students are invited.

Friday, November 8, 12-1, Cheever Amphitheatre. Dr. William R. Morrison, Associate Professor of Surgery, Boston University School of Medicine, will present the following cases:

1. Ileosigmoidostomy.
2. Resection of Stomach for Adenocarcinoma, Using Von Petz Sewing Clamps.
3. Femoral Hernia.

Physicians and medical students are invited.

BOSTON DISPENSARY

ASSEMBLY HALL, 25 BENNET STREET

Medical Conference Program
9-10 A.M., November, 1935

November 1, Friday—Ward Cases. Dr. S. J. Thannhauser.

November 2, Saturday—Diabetes. Dr. S. J. Thannhauser.

November 4, Monday—Nephritic Clinic. Dr. R. W. Buck.

November 5, Tuesday—"Diagnosis of Spinal Conditions." Dr. J. D. Adams.

November 6, Wednesday—Ward Cases. Dr. S. J. Thannhauser.

November 7, Thursday—"Revision of Interpretation of Laboratory Tests for Syphilis." (Continued.) Dr. W. A. Hinton.

November 8, Friday—Ward Cases. Dr. S. J. Thannhauser.

November 9, Saturday—Diabetes. (Continued.) Dr. S. J. Thannhauser.

November 12, Tuesday—"Report on the Results of Short-Wave Therapy in the Past Year." Dr. P. Hoefer.

November 13, Wednesday—Ward Cases. Dr. S. J. Thannhauser.

November 14, Thursday—"Recent Observations on the Functional Properties of the Vascular System and on the Hemodynamics in Arterial Hypertension." Dr. Soma Weiss.

November 15, Friday—Ward Cases. Dr. S. J. Thannhauser.

November 16, Saturday—Liver Disease. Dr. S. J. Thannhauser.

November 18, Monday—Blood Clinic. Dr. William Dameshek.

November 19, Tuesday—Back Strain—Sciatica. Dr. F. R. Ober.

November 20, Wednesday—Ward Cases. Dr. S. J. Thannhauser.

November 21, Thursday — "Interviewing a Patient." Prof. Elton Mayo.

November 22, Friday—Ward Cases. Dr. S. J. Thannhauser.

November 23, Saturday—Liver Disease. (Continued.) Dr. S. J. Thannhauser.

November 25, Monday—Heart Clinic. Dr. S. H. Proger and Mrs. C. Janeway.

November 26, Tuesday—X-Ray Demonstration. Dr. A. Ettinger.

November 27, Wednesday—Ward Cases. Dr. S. J. Thannhauser.

November 29, Friday—Ward Cases. Dr. S. J. Thannhauser.

November 30, Saturday — Peptic Ulcer. Dr. S. J. Thannhauser.

HARVARD MEDICAL SCHOOL

THE GEORGE W. GAY LECTURE ON "MEDICAL ETHICS"

Amphitheatre C at 5 P.M.

Thursday, November 7—Dr. James B. Herrick of Chicago.

LECTURES ON "THE CARE OF THE PATIENT"

Amphitheatre C at 5 P.M.

Thursday, November 14—Dr. Arthur R. Crandell of Taunton.

Thursday, November 21—Dr. David D. Scannell of Boston.

FAULKNER HOSPITAL CLINICAL MEETING

The next clinical meeting at the Faulkner Hospital will be held on Thursday, November 7, at 5:00 P.M. In addition to the usual clinical pathological conference Dr. Chester M. Jones will talk on "Enteritis, Symptomatology and Diagnosis."

All physicians are invited.

ESSEX SOUTH DISTRICT MEDICAL SOCIETY

The regular meeting of the Essex South District Medical Society will be held Wednesday, November 6, 1935, at the Beverly Hospital.

Clinic at 5 P.M. Dinner at 7 P.M.

Speaker: Dr. Charles E. Mongan, of Somerville, President of the Massachusetts Medical Society, who will talk on "Matters of Interest to the Medical Profession."

HANFORD CARVELL, M.D., *President*,
R. E. STONE, M.D., *Secretary*.

BARNSTABLE DISTRICT MEDICAL SOCIETY

THURSDAY, NOVEMBER 7, 1935

Business Dinner, 12:30 at Bill Cox's Sea Grill, Hyannis Park.

Scientific Meeting, 2-4 P.M. at Cape Cod Hospital.

Dr. Henry Pinkerton—Results of Tumor Research at Falmouth Institute.

Mr. Walter Hughes—The Sex Factor in Cancer.

Dr. Samuel M. Beale—Clinical Experiences.

Discussion opened by Dr. Shields Warren.

J. I. B. VAIL, M.D., *Secretary*.

WILLIAM HARVEY SOCIETY

The next meeting of the William Harvey Society will be held Friday, November 8, in the Auditorium of the Beth Israel Hospital, Boston, at 8:00 P.M.

PROGRAM

Speaker: Dr. Harold E. B. Pardee, Assistant Professor of Clinical Medicine, Cornell University Medical School.

Subject: "Arteriosclerotic Heart Disease."

Chairman: Dr. Cadis Phipps, Professor of Medicine, Tufts College Medical School.

THE ATLANTIC DERMATOLOGICAL CONFERENCE

This meeting will be held in Boston, Mass., Saturday, November 9, 1935.

Clinical Meeting at the Boston City Hospital:

1:30 P.M. Luncheon at the Boston City Hospital in complement to the conference by the Board of Trustees.

2:15 P.M. Demonstration of 30 cases.

4:00 P.M. Discussion of the cases with complete freedom of thought guaranteed under the Constitution of the Conference, which is revered by all but has been preserved by none.

Dinner at the Hotel Kenmore, 490 Commonwealth Avenue:

7:00 P.M. "Be large in mirth; anon we'll drink a measure the table round." Macbeth, III, 4.

7:30 P.M. "Sit down and feed, and welcome to our table." As You Like It, II, 7.

Newell Bent, Jr., Explorer, Naturalist and Photographer, will show motion pictures and chat of his trip which he made alone from Cape Town to Cairo. He climbed the snow-capped volcano, Killimarjaro; he hunted big game with his camera; and he consorted at times with cannibals.

The conference is limited strictly to its members.

J. HARPER BLAISDELL, M.D., *Secretary*.

HARVARD MEDICAL SOCIETY

The next meeting of the Harvard Medical Society will be held in the Peter Bent Brigham Hospital Amphitheatre (Shattuck Street Entrance), Tuesday evening, November 12, at 8:15 P.M.

PROGRAM

Presentation of Cases.

Recent Studies in Yellow Fever—A Virus Disease. By Dr. Frederick F. Russell, Lecturer in Preventive Medicine and Hygiene, Harvard Medical School.

MARSHALL N. FULTON, M.D., *Secretary.*

SOCIETY MEETINGS, CONGRESSES AND CONFERENCES

CALENDAR OF BOSTON DISTRICT FOR THE WEEK BEGINNING MONDAY, NOVEMBER 4, 1935

Monday, November 4—

*9-10 A.M. Boston Dispensary, 25 Bennet Street, Boston. Nephritic Clinic. Dr. R. W. Buck.

Tuesday, November 5—

*9-10 A.M. Boston Dispensary, 25 Bennet Street, Boston. "Diagnosis of Spinal Conditions." Dr. J. D. Adams.

2:30 P.M. Pediatric Ward Visit. Massachusetts Eye and Ear Infirmary.

7:45 P.M. Gardner Auditorium, State House, Boston. "Keeping Mentally Fit." Dr. Joseph Jastrow.

Wednesday, November 6—

*9-10 A.M. Boston Dispensary, 25 Bennet Street, Boston. Ward Cases. Dr. S. J. Thannhauser.

†12 M. Clinico-Pathological Conference. Children's Hospital.

Thursday, November 7—

*8:30-9:30 A.M. Clinic, Surgical Staff of the Peter Bent Brigham Hospital, at the Peter Bent Brigham Hospital.

*9-10 A.M. Boston Dispensary, 25 Bennet Street, Boston. "Revision of Interpretation of Laboratory Tests for Syphilis" (Cont'd). Dr. W. A. Hinton.

*3:30 P.M. Medical Clinic at the Peter Bent Brigham Hospital.

5 P.M. Clinical Meeting, Faulkner Hospital.

5 P.M. Harvard Medical School. The George W. Gay Lecture on "Medical Ethics."

Friday, November 8—

*9-10 A.M. Boston Dispensary, 25 Bennet Street, Boston. Ward Cases. Dr. S. J. Thannhauser.

12 M. Clinical Meeting of the Children's Medical Staff. Ether Dome, Massachusetts General Hospital.

*12-1. Boston University School of Medicine Surgical Clinic at the Boston City Hospital (Cheever Amphitheatre).

8 P.M. William Harvey Society. Auditorium, Beth Israel Hospital, Boston.

Saturday, November 9—

*9-10 A.M. Boston Dispensary, 25 Bennet Street, Boston. Diabetes (Cont'd). Dr. S. J. Thannhauser.

*10-12. Staff rounds at the Peter Bent Brigham Hospital.

1:30-4 P.M. The Atlantic Dermatological Conference, Boston City Hospital. 7 P.M. Hotel Kenmore.

*Open to the medical profession.
†Open to Fellows of the Massachusetts Medical Society.

November 1—Boston University School of Medicine Surgical Clinic at the Boston City Hospital. See page 890.

November 1-30—Boston Dispensary, Medical Conference Program. See page 890.

November 2, 9, 16, 23—Lectures by Dr. S. J. Thannhauser. See page 889.

November 5 - January 14—Massachusetts Department of Education, Mental Hygiene for Adults. See page 890.

November 7—Medical Clinic at the Peter Bent Brigham Hospital. See page 889.

November 7—Harvard Medical School. The George W. Gay Lecture on "Medical Ethics." See page 891.

November 7—Faulkner Hospital Clinical Meeting. See page 891.

November 8—William Harvey Society. See page 891.

November 8—Boston University School of Medicine Surgical Clinic at the Boston City Hospital. See page 890.

November 9—The Atlantic Dermatological Conference. See page 891.

November 12—Harvard Medical Society. See notice elsewhere on this page.

November 14—Harvard Medical School. Lecture on "The Care of the Patient." See page 891.

November 21—Harvard Medical School. Lecture on "The Care of the Patient." See page 891.

DISTRICT MEDICAL SOCIETIES

BARNSTABLE DISTRICT MEDICAL SOCIETY

November 7—See page 891.

ESSEX NORTH DISTRICT MEDICAL SOCIETY

November 7—Censors' Meeting at the Hotel Bartlett, Main Street, Haverhill, at 4 P.M.

ESSEX SOUTH DISTRICT MEDICAL SOCIETY

November 6—See page 891.

FRANKLIN DISTRICT MEDICAL SOCIETY

Meetings are held on the second Tuesday of November, January, March and May at the Weldon Hotel, Greenfield, at 11 A.M.

CHARLES MOLINE, M.D., Secretary.

HAMPDEN DISTRICT MEDICAL SOCIETY

November 7—Censors' Meeting in the rooms of the Academy of Medicine, 20 Maple Street, Springfield, at 4 P.M.

SUFFOLK DISTRICT MEDICAL SOCIETY

November 7—Censors' Meeting will be held at the Boston Medical Library, 8 Fenway, at 4 P.M.

December 11—Joint Meeting with the New England Heart Association at the Boston Medical Library. "Constrictive Disease of the Pericardium," Dr. Charles Sidney Burwell. Discussion: Dr. Edward D. Churchill and Dr. Paul D. White.

January 29, 1936—Joint Meeting with the Boston Medical Library at 8 Fenway. "Observations Around the World," Dr. Walter B. Cannon.

March 18, 1936—Meeting at the Boston Medical Library. "The Laboratory and Clinical Story of Fatigue," Dr. Arlie V. Bock and Dr. David B. Dill. Discussion: Dr. Donald J. McPherson and Dr. Augustus Thorndike, Jr.

April 29, 1936—Annual Meeting at the Boston Medical Library. "The Treatment of Septicaemia," Dr. Champ Lyons. "The Pleurality of Scarlatinal Streptococcus Toxin," Dr. Sanford B. Hooker. Discussion: Dr. Hans Zinsser.

The medical profession is cordially invited to attend all of these meetings.

ROBERT L. DeNORMANDIE, M.D., President,
CHARLES C. LUND, M.D., Secretary,
FRANCIS T. HUNTER, M.D.,
Boston Medical Library.

WORCESTER DISTRICT MEDICAL SOCIETY

November 7—Censors' Meeting will be held in the Library Rooms of the Worcester District Medical Library, Inc., 34 Elm Street, Worcester, at 4:30 P.M.

November 13—Wednesday evening. Grafton State Hospital, North Grafton, Mass. Dinner and scientific program. Subjects of program to be announced later.

December 11—Wednesday evening. St. Vincent Hospital, Worcester, Mass. Dinner and scientific program. Subjects of program to be announced later.

January 8, 1936—Wednesday evening. Worcester City Hospital, Worcester, Mass. Dinner and scientific program. Subjects of program to be announced later.

February 12, 1936—Wednesday evening. Worcester State Hospital, Worcester, Mass. Dinner and scientific program. Subjects of program to be announced later.

March 11, 1936—Wednesday evening. Memorial Hospital, Worcester, Mass. Dinner and scientific program. Subjects of program to be announced later.

April 8, 1936—Wednesday evening. Hahnemann Hospital, Worcester, Mass. Dinner and scientific program. Subjects of program to be announced later.

May 13, 1936—Wednesday afternoon and evening. Annual Meeting of Society. Time, place and details of program to be announced in an April issue of the Journal.

ERWIN C. MILLER, M.D., Secretary.